THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—17TH YEAR. SYDNEY, SATURDAY, SEPTEMBER 13, 1930. No. 11.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.

"Local Venesection in the Treatment of Snake Bite: An Experimental Study," by C. H. KELLAWAY, M.C., M.D., M.S., F.R.C.P. 351

"The Influence of Extracts of Prickly Pear on Diabetes Mellitus," by W. WILSON INGRAM, M.C., M.D., and G. V. RUDD, M.Sc. 360

"The White Cedar or Azedarach," by JOHN MACPHERSON, M.A., B.Sc., M.B., Ch.M. 363

"The Treatment of Blindness Associated with Retinitis Pigmentosa: A Preliminary Note," by N. D. ROYLE, M.D., Ch.M., F.C.S.A. 364

REPORTS OF CASES—

"Cerebro-Spinal Fever," by W. E. HASKER, M.B., B.S. 365

"The Treatment of Deafness by the Zund-Burguet Method," by R. H. Bettington, B.A., B.M., B.Ch. 366

REVIEWS—

The Tonsils and Tonsillar Surgery 367

Ophthalmology 368

A Résumé of Obstetrics and Gynæcology 368

LEADING ARTICLES—

Research and the Community 369

CURRENT COMMENT—

Hyperparathyreoidism 370

Carcinoma of the Duodenum 371

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Gynæcology 372

Obstetrics 372

SPECIAL ARTICLES ON DIAGNOSIS—

Precordial Pain 374

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 375

The Jubilee of the New South Wales Branch .. 378

NAVAL, MILITARY AND AIR FORCE—

Appointments 378

CORRESPONDENCE—

Otitis Media 379

NOTICE 380

BOOKS RECEIVED 380

DIARY FOR THE MONTH 380

MEDICAL APPOINTMENTS 380

MEDICAL APPOINTMENTS VACANT, ETC. 380

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 380

EDITORIAL NOTICES 380

LOCAL VENESECTION IN THE TREATMENT OF SNAKE BITE: AN EXPERIMENTAL STUDY.[1]

By C. H. KELLAWAY, M.C., M.D., M.S. (Melbourne), F.R.C.P. (London).

(From the Walter and Eliza Hall Institute, Melbourne.)

HAMILTON FAIRLEY'S 1929 recent work[(1)] has shaken our faith in the value of ligature alone as a remedy for snake bite, except in the sense that it delays the entry of venom into the general circulation and affords time for the application of other treatment. For these reasons ligature applied round a part of the limb which contains a single bone, in order to obstruct completely the arterial flow to the ligated part, must remain the first and most important step in the treatment of snake bite.

Martin's[(2)(3)] experiments on the rabbit using the venom of the black snake (*Pseudechis porphyriacus*) and tiger snake (*Notechis scutatus*) had shown that in this species simple ligature even for so short a period as twenty minutes was effective in saving life, even against six to ten certainly lethal doses of the first-mentioned venom, but that if heated venom were used the only observed effect was prolongation of the death time. The usefulness of ligature in the treatment of snake bite in the rabbit was thus shown to depend on the heat-labile constituent of the venom, that is on the thrombase —the venom being retained in the tissues of the bitten part owing to local thrombosis. The work of Acton and Knowles[(4)] had shown that ligature was useful in the treatment of bites by the daboia (a thrombase containing venom), but had demonstrated that its only effect in bites by the cobra (whose venom contains no thrombase but instead an anticoagulant

[1] This research was carried out under a grant from the Commonwealth Government Department of Health.

factor) is a delay in the death time. In Fairley's experiments on goats and sheep the venoms of the tiger snake (*Notechis scutatus*), death adder (*Acanthophis antarcticus*) and copper-head (*Denisonia superba*) (the last two venoms being feebly anticoagulant), were used. Simple ligature was found to be wholly without effect in the treatment of both species of experimental animal, not saving the animal against even a single certainly lethal dose of these venoms. Fairley explained the difference between his results with tiger snake venom and those of Martin by the very much less powerful thrombase action of this venom in the sheep as compared with its action in the rabbit. Experiments were also made on the treatment of sheep after subcutaneous injection of the venoms of the cobra (*Naia naia*) and the daboia (*Vipera russelli*). Ligature appeared to be of some use in the treatment of sheep injected with the latter.

Acton and Knowles[4] had shown that amputation of the tails of monkeys into which ten certainly lethal doses of cobra venom had been injected subcutaneously, was effective in saving life if carried out within six minutes. Fairley[5] made the important observation that sheep bitten naturally by tiger snakes survived if an effective ligature were applied and the area of the bite widely and freely excised immediately. This appears to be the only local treatment in bites by the Australian venomous snakes whose value has been demonstrated, and its usefulness depends on the promptness with which it is applied.

There can be no doubt that in the case of some at least of the Australian elapine venoms when these are injected subcutaneously, absorption is directly into the bloodstream and not by way of the lymphatics. Martin[6] showed that ligature of the thoracic duct did not cause any delay in the onset of symptoms, following the subcutaneous injection of the venom of the black snake, and the experiments recorded in the present paper strongly support the view that the main channel of entry of the Australian venoms into the body is by way of the bloodstream and not by the lymphatics or by tissue spread. When the venom is administered subcutaneously, the rapidity with which a lethal dose enters the general circulation, depends upon the size of the dose. With doses of about the same size as the certainly lethal dose the onset of symptoms indicates that the absorption of the whole of the venom injected takes some hours. Following the subcutaneous injection of very large doses the absorption of a single certainly lethal dose may take place within a few minutes, hence the vital necessity for immediate application of ligature. I have recently described[7] a new method for the treatment of snake bite of the limbs which depends upon the application of two ligatures: (i) an "arterial" ligature applied round a part of the limb which contains only one bone (for example, the arm or thigh), and (ii) immediately distal to this, a "venous" ligature which theoretically should be tight enough to obstruct the venous return from the bitten part, but not tight enough to prevent the entry of arterial blood whenever the proximal "arterial" ligature is removed. The essence of the treatment lies in the removal by venesection of envenomed venous blood, from the part distal to the "venous" ligature. A number of bleedings are carried out, the "arterial" ligature being removed for about a minute or a little more and reapplied after each bleeding. If much of the venom normally diffuses into the bloodstream within a short time of the bite and its portal of entry to the body is the venous return from the bitten part, a large proportion of it should be removed in the successive fractions of venous blood which are collected. Under these conditions, if the dose of venom injected is not much in excess of a lethal one, this treatment alone should be successful in reducing to a sublethal dose the amount of venom finally entering the general circulation. If, however, the venom normally gains access but slowly to the general circulation and has a strong affinity for the tissues surrounding the area of the bite or if absorption is by the lymphatics, not much can be expected of the treatment. The period during which the "arterial" ligature can be repeatedly taken off and reapplied is obviously limited by the local bruising and damage to the tissues at the site of the ligature and by the amount of blood which can safely be let. In children and in adults who have received a large dose, the number of bleedings carried out can be increased by the transfusion of blood from a normal individual.

The experiments presented here were made on sheep, since the lethal doses of the common venoms have been worked out for this animal by Fairley. The general method followed was to apply the "arterial" and "venous" ligatures to a limb and then to inject the venom and proceed with treatment by venesection only. As an "arterial" ligature a rubber tourniquet applied on the stretch was placed round the left forelimb above the carpal joint. In Fairley's experiments, the ligature was applied below the carpal joint round the metacarpal bone, but in the present experiments it was desired to have a greater bulk of tissue distal to the ligature. By placing the ligature just above the carpal joint it was virtually round a single bone, for the primitive ulna is fused with the radius in its lower portion. In practice we found that as a rule only a trivial amount of blood entered the ligated part when the "arterial" ligature was in position, though in one or two animals even a very tight ligature in this position failed to obstruct so completely the inflow of arterial blood. The "venous" ligature which was a narrow cotton bandage, was applied immediately distal to the "arterial" ligature. The venom was then injected subcutaneously in the vicinity of the metacarpophalangeal articulation in concentrations identical with those used by Fairley in his experiments, so as to allow of the utilization of his ligature experiments as controls. An incision was made into a suitable subcutaneous vein immediately behind the carpal joint and at intervals of five minutes for about an hour the "arterial" ligature was taken off for one to one and a half minutes. During these short periods the blood flowed freely from the venesection wound and was caught in a measure glass containing

180 cubic centimetres of 2% sodium citrate in normal saline solution. When a quantity of blood equal in amount to the citrate solution had been obtained (usually about 30 cubic centimetres for each removal of the "arterial" ligature) a fresh measure containing 180 cubic centimetres of the citrate saline solution was used for collection. In most of the experiments a total of 280 cubic centimetres to 400 cubic centimetres of blood was removed. With the larger sheep even greater amounts were bled and no serious effects from hæmorrhage were observed. When coagulant venoms had been injected (for example, those of the tiger snake and of the common brown snake) it was always necessary to tie the vein (after the ligature was finally removed) to prevent continued bleeding of "negative phase" blood.

The first step in the investigation of the value of the method was to ascertain whether the blood removed by local venesection really contained any appreciable quantity of venom. Two experiments were made by transferring the whole of the blood removed from one sheep into another and for economy other confirmatory tests were carried out upon guinea-pigs. The experiments in which sheep were injected with venesection blood were made with death adder venom, since for obvious reasons a venom containing thrombase was not suitable. (The blood removed from sheep injected with tiger snake and brown snake venoms regularly clotted despite its admixture with citrate.) The two sheep, the donor and recipient, were tested for possible cross agglutination before the experiment was made.

EXPERIMENT I.—A sheep weighing 42·7 kilograms received subcutaneously three certainly lethal doses of death adder venom (3·2 milligrammes in 0·8 cubic centimetre) and subsequently 200 cubic centimetres of blood were allowed to flow from the venesection wound into an equal quantity of sterile saline solution containing 2% citrate. The arterial ligature was finally removed after one hour, during the course of which ten successive bleedings had been carried out. This animal died in less than seventeen hours. The citrated blood from this sheep (400 cubic centimetres) was filtered through glass wool and run slowly by gravity into the jugular vein of a second sheep weighing 20 kilograms. This second sheep likewise died in about fifteen hours with symptoms suggestive of poisoning by death adder venom. *Post mortem* there was no clotting in the second animal and the autopsy findings were identical with those of the treated sheep and consistent with death by poisoning with death adder venom.

In the second experiment the effect of transfusion was studied. The two sheep used—Numbers 62 and 64—were tested for possible cross agglutination with negative results. A full protocol of this experiment is given.

EXPERIMENT II (May 22, 1930).—To sheep 64, a ewe, weighing 40·3 kilograms, "venous" and "arterial" ligatures were applied on the left forelimb above the carpal joint. Two certainly lethal doses of death adder venom (2·0 milligrammes in 0·5 cubic centimetre of saline solution) were injected subcutaneously over the metacarpo-phalangeal joint at 12.4 p.m. Seven successive bleedings were made at 12.10, 12.14, 12.19, 12.26, 12.30, 12.35 and 12.42 p.m., a total of 280 cubic centimetres being bled into an equal volume of citrate. From sheep 62, 220 cubic centimetres of blood were now drawn from a jugular vein into an equal quantity of saline solution containing 2% sodium citrate. This citrated blood, warmed to body temperature, was now allowed to flow slowly into a jugular vein of sheep 64, twenty-five minutes being allowed for the transfusion, which was complete at 1.55 p.m. A second series of eight bleedings were now made from sheep 64, at 1.58, 2.3, 2.8, 2.13, 2.18, 2.28, 2.37 and 2.42 p.m., a total of 270 cubic centimetres of envenomed blood being removed. The sheep had now been bled altogether 550 cubic centimetres and had received by transfusion 440 cubic centimetres of citrated blood. The ligatures were removed at 2.44 p.m. Ten hours after the injection the animal became sick and regurgitated fluid from its rumen. After twenty-one hours the animal had a wet nose and seemed somewhat weak and two hours later though there was some frothing from the nose and mouth and severe dyspnœa, the animal was not paralysed. After twenty-eight hours the sheep was much worse and though it was "down" there was still no paralysis of the limbs. It showed no improvement and died at 12.20 p.m. on May 24, 1930, approximately forty-eight hours after the injection. *Post mortem* the blood was fluid except for a *post mortem* clot in the heart. The lungs were intensely congested and there was a hæmorrhage in the right suprarenal gland. The kidneys and the other suprarenal gland were congested and the spleen congested and hæmorrhagic.

Sheep 62 was a wether weighing 34·4 kilograms. From 12.50 to 1.15 p.m. it was bled 220 cubic centimetres from the right jugular vein. From 2.55 to 3.15 p.m. 780 cubic centimetres of warmed citrated blood from sheep 64 were slowly run into the left jugular vein. The remainder of the blood removed from sheep 64 was divided into two equal parts and run by gravity under the loose skin of the inside of the thigh and lower part of the abdominal wall on both sides. Within eight and a quarter hours the animal was profoundly paralysed. It was regurgitating fluid from its rumen, its nose and mouth were wet and it had severe dyspnœa. Death took place at 3.15 p.m., May 23, 1930, about twenty-four hours after the injection of envenomed blood. *Post mortem* the subcutaneously injected fluid had been absorbed. There was no œdema or free fluid in the body cavities. The blood everywhere was fluid. The lungs were extensively collapsed with large areas of hæmorrhage and there were a few hæmorrhages elsewhere. The kidneys were intensely congested and so also was the bladder. There was no free blood or pigment in the urine.

It is certain that this second sheep died from the injection of death adder venom, and the result of the experiment suggests that about half the venom had been removed from sheep 64 by the treatment though not quite enough to save it. It will be seen later that without transfusion I was able to save sheep which had received one and a quarter and one and a half, but not one and three-quarters certainly lethal doses of this venom.

Since this type of experiment was costly, other experiments were made in which guinea-pigs weighing about 250 grammes were used for testing for the presence of venom in the citrated blood obtained by venesection. The certainly lethal doses of the venoms given subcutaneously to guinea-pigs of this weight are 0·002 milligramme per 100 grammes for tiger snake venom, rather more than 0·006 milligramme per 100 grammes for copper-head venom and 0·015 milligramme per 100 grammes for the venom of the death adder (Kellaway[8][9][10]). The guinea-pigs received citrated blood from sheep into which one and a half or one and a quarter certainly lethal doses of the venoms of the copper-head or death adder had been injected. In the case of tiger snake venom the diluted serum which exuded from the clotted blood was used. It was found possible to inject twenty cubic centimetres of normal citrated sheep blood or plasma into guinea-pigs without causing more than a temporary malaise, if the injections were divided and made under the skin of the abdominal wall and thorax in a number of positions. Guinea-pigs injected with the citrated serum from the local venesection of sheep injected with tiger snake venom, invariably

succumbed with doses of 12 to 15 cubic centimetres of serum, but even with doses of 20 cubic centimetres of the blood from sheep treated following the injection of copper-head or death adder venom the guinea-pigs survived.

Into a sheep weighing 40·5 kilograms one and a quarter certainly lethal doses (0·51 milligramme) of tiger snake venom were injected subcutaneously and during an hour's treatment 280 cubic centimetres of blood were bled into an equal quantity of citrate saline solution. Four guinea-pigs weighing 287, 250, 280 and 230 grammes received 20, 15, 12 and 10 cubic centimetres of the serum. All exhibited typical neurotoxic symptoms. The first died in twelve hours, the second on the second day, the third on the third day and the last survived.

In this experiment the first two guinea-pigs received serum from blood collected during the first half hour of the venesection and the second pair, serum from blood obtained during the second half hour. Obviously the blood removed in each successive bleeding must contain progressively less venom, but even during the second half of the first hour an appreciable amount of tiger snake venom was being removed. Assuming that the serum administered was approximately half saline solution and that the venom was equally distributed between the serum and the clot, the blood removed by venesection must have contained approximately 0·001 milligramme of venom per cubic centimetre and the total dose received by the sheep (0·51 milligramme) must have been reduced to about half by the treatment. The sheep survived without showing any symptoms.

In another experiment a sheep weighing 40 kilograms received one and a half certainly lethal doses (0·6 milligramme) of tiger snake venom. During just over an hour's treatment it was bled 340 cubic centimetres. Guinea-pigs weighing 235 and 320 grammes received 15 cubic centimetres each of the serum diluted with citrate obtained during the first half hour of bleeding. Both died with typical neurotoxic symptoms in less than thirty-six hours. The sheep was very severely paralysed after twenty-four hours and remained so for five days. After recovery had apparently commenced it relapsed two days later and as its general condition was rather miserable, it was killed.

In this case during the first half hour of the treatment (170 cubic centimetres × 0·0008 milligramme) 0·136 milligramme of venom must have been removed, and even if during the second half of the treatment only half this amount was eliminated the effective dose must have been reduced by about one-third. The severity of the symptoms are not surprising, since Fairley's experiments show that occasional sheep are killed by doses of 0·006 and 0·007 milligramme per kilogram (that is 0·24 to 0·28 milligramme total dose for a sheep of this size).

In contrast to these results were those obtained by the injection of doses of 20 cubic centimetres of citrated blood from sheep injected with the venom of the death adder and of the copper-head.

For example, two guinea-pigs weighing 300 and 270 grammes respectively received 20 and 18 cubic centimetres of blood (total venesection 320 cubic centimetres) from a sheep of 34 kilograms which had been injected with one and a half lethal doses (1·26 milligrammes total dose) of death adder venom. Both of these survived without symptoms as also did the sheep from which the blood was taken.

Similarly two guinea-pigs weighing 230 and 245 grammes survived without symptoms after being injected with 18 cubic centimetres each of the blood (total venesection 280 cubic centimetres) from a sheep weighing 30 kilograms which received one and a fifth lethal doses (total dose 3·6 milligrammes) of copper-head venom. This sheep recovered after being paralysed for six days.

The result in the case of guinea-pigs injected with blood from the death adder sheep was to be expected even assuming that one-third of the injected venom had been removed by treatment. The blood on this assumption would contain about 0·0013 milligramme per cubic centimetre and 20 cubic centimetres of the citrated blood would contain therefore approximately 0·013 milligramme or about one-third of a certainly lethal dose for a guinea-pig weighing 300 grammes. In the case of the blood from the sheep injected with copper-head venom, on the assumption that one-third of the venom was removed, the diluted citrated blood would contain about 0·002 milligramme of venom per cubic centimetre and 18 cubic centimetres should have contained 0·036 milligramme of copper-head, an amount nearly sufficient to kill two guinea-pigs of 250 grammes weight. It is probable therefore that in the case of this venom the amount removed during the course of one hour was less than one-sixth of the total amount of venom injected into the sheep.

These results and those of other similar experiments suggest that the method is probably efficacious for treatment after injection of the venoms of the death adder and tiger snake, but relatively ineffective after the injection of copper-head venom.

The Results of Treatment after the Injection of the Venom of the Death Adder (Acanthophis antarcticus).

The results of treatment after the injection of the venom of the death adder, *Acanthophis antarcticus*, are set out in Table I. From consideration of their cost only a limited number of experiments have been made. They are weighted in favour of recovery by the fact that the ligatures were applied immediately before the injection of the venom, but on the other hand the "arterial" ligature applied above the carpal joint did not always completely obstruct the inflow of arterial blood to the ligated part, nor was the venous ligature theoretically perfect. Doubtless there was some leak of envenomed venous blood into the general circulation when the "arterial" ligature was in position and even more during the periods in which the ligature was removed for bleeding. This was done for about one minute or a little longer in every five minutes during the course of about one hour. It always required some seconds after the removal of the ligature for the ligated part to fill up with blood and before the flow of blood from the venesection wound commenced. This point has already been stressed by Fairley in discussing the value of removing the ligature at intervals to avoid damage to the ligated part.

The venom in these experiments was injected in a concentration of 4 milligrammes per cubic centimetre. Fairley, using this concentration, applied simple ligature below the carpal joint to the treatment of four sheep which weighed 43·8, 44·3, 44·5 and 57·3 kilograms and which received subcutaneously 1·1, 1·2, 1·3 and 3·4 milligrammes of venom (1·0, 1·1, 1·2 and 2·4 lethal doses). These animals

died in 44½ hours, 22 hours, 12 hours and 5¼ hours respectively. The method under investigation is therefore very much more effective than is simple ligature.

TABLE I.

The Effect of Treatment After the Injection of Death Adder Venom.

Weight of Sheep in Kilograms.	Number of Centainly Lethal Doses Injected.	Total Dose of Venom in Milligrammes.	Amount of Blood Removed by Venesection in Cubic Centimetres.	Result.
42·7	3	3·2	200	Death in less than seventeen hours.
18	2	0·9	170	Died in less than twenty-two hours.
40·3	2	2·0	550	Died in forty-eight hours.
45·3	1¾	1·98	400	Died in ten hours.
34	1½	1·27	350	Survived without symptoms.
31·5	1½	1·18	310	Only slight ataxia and weakness on the third day; recovered by fourth day.
23	1¼	0·72	110	Died on fourth day.

When however, we are concerned with the saving effect against doses only half as much again as the lethal dose, possible inaccuracy in the determination of this dose may be important. Fairley's estimated certainly lethal dose for death adder venom in sheep, 0·025 milligramme per kilogram, rests upon the fatal effects of this dose in three animals which died in 45½, 28⅔ and 44⅓ hours respectively. With only slightly larger dosage—0·0275 and 0·03 milligramme per kilogram—the death time was shortened to 14½ and 11 hours respectively, and it is therefore likely that these last two sheep received much more than the certainly lethal dose. For Fairley's purpose a greater number of animals was not necessary, since, if the lethal dose were higher than that indicated by the results in his necessarily limited number of animals, any possible underestimation of the value was not sufficient to operate favourably in his ligature experiments. In the present experiments the difference between one and a half certainly lethal doses—0·037—and one certainly lethal dose—0·025 milligramme per kilogram—is too great to be affected by any small divergence of the actual certainly lethal dose from that estimated by Fairley. It will be noted that one two-tooth sheep weighing 23 kilograms died on the fourth day after the injection of only one and a quarter certainly lethal doses of venom. This animal was in such poor condition that it was only bled 110 cubic centimetres, the ligature being finally removed after forty minutes. It is not therefore a fair test of the efficacy of the method.

The Results of Treatment after the Injection of Tiger Snake (Notechis scutatus) Venom.

In Table II are set out the results of treatment after injection of tiger snake venom. The venom was administered in a concentration of one milligramme per cubic centimetre (that used in Fairley's ligature experiments). The number of bleedings varied from ten to twelve and the ligatures were finally removed after about an hour.

TABLE II.

The Effect of Treatment After the Injection of Tiger Snake Venom.

Weight of Sheep in Kilograms.	Number of Certainly Lethal Doses Injected.	Total Dose in Milligrammes.	Amount of Blood Removed by Venesection in Cubic Centimetres.	Result.
50·7	1·5	0·76	390	Weakness in the hind limbs on the second day, paralysed from the third to the seventh day; recovery.
40	1·5	0·1	360	Paralysed after twenty-four hours and remained so for five days, after which it was able to stand and take food and drink; it relapsed two days later and was killed on the eighth day.
36·7	1·5	0·55	370	Weakness and ataxia after twenty-seven hours, paralysed from the end of the second to the thirteenth day; recovery.
40·5	1·25	0·51	280	Survived without symptoms.

It will be observed that in the case of this venom recovery without symptoms occurred in the sheep which received one and a quarter certainly lethal doses, but after one and a half certainly lethal doses all the sheep had symptoms which indicated that the dose of venom absorbed was only just sublethal. With careful nursing the second sheep which received one and a half certainly lethal doses should have recovered. It was severely affected with caseous lymphadenitis and was in extremely poor condition before the experiment. When it relapsed after apparently recovering completely, it was killed. No lesions suggestive of tiger snake poisoning were found at autopsy.

The observations in guinea-pigs killed by the injection of serum obtained in the course of these experiments and the results of the experiments themselves demonstrate the value of the method, especially when these last results are compared with those obtained by Fairley in experiments on simple ligature. In these, four sheep weighing 34·7, 46·3, 46·4 and 46·8 kilograms were injected with one certainly lethal dose, one weighing 41·4 kilograms received one and a quarter certainly lethal doses and one weighing 33·9 kilograms received one and a half certainly lethal doses. All these animals died within twenty-nine hours after the injection. In view of these last results, Fairley's estimation of the certainly lethal dose (0·01 milligramme per kilogram) cannot be far removed from the true figure.

The Results of Treatment in Sheep Injected with the Venom of the Copper-Head (Denisonia superba).

Experiments dealing with the results of treatment with the venom of the copper-head, *Denisonia*

superba, were by far the least satisfactory of the present experiments. In view of the small number of animals upon which Fairley's determination of the certainly lethal dose was made, I injected a few sheep with a dose somewhat smaller than his estimated certainly lethal dose (0·1 milligramme per kilogram) in order to make certain that this figure was not too high. The results in these sheep are set out in Table III. The venom was administered in a concentration of 10 milligrammes per cubic centimetre.

TABLE III.

Observations on the Certainly Lethal Dose of the Venom of the Copper-head (Denisonia superba) Injected Subcutaneously into Sheep.

Weight in Kilograms.	Total Dosage in Milligrammes.	Dose in Milligrammes per Kilogram.	Result.
55·3	4·42	0·08	Paralysed after twelve hours, died in twenty-two hours.
43·9	3·5	0·08	Paralysed after twelve hours, died in twenty-four hours.
36	2·9	0·08	Died in twenty-three hours.
49·1	3·9	0·08	Paralysed by the end of the first day and not completely recovered after twenty-two days.
48·9	3·9	0·08	Paralysed by the end of the first day and recovered by the twelfth day.

In Fairley's series a sheep which received 0·07 milligramme per kilogram recovered and two which received 0·10 milligramme per kilogram died after twenty-four and twenty-five hours respectively. Another animal which received this last dose and was treated by simple ligature recovered after being paralysed for two days. The observations recorded in Table III make it clear that a dose of 0·08 milligramme is just sublethal, the two sheep which recovered having been paralysed for long periods. Taking all the evidence together Fairley's estimated dose, 0·1 milligramme per kilogram, cannot be far from the truth and has therefore been taken as the approximate certainly lethal dose. The results of treatment by local venesection after injection of this venom are set out in Table IV.

TABLE IV.

Results of Treatment in Sheep Injected with the Venom of the Copper-head.

Weight of Sheep in Kilograms.	Number of Certainly Lethal Doses Injected.	Total Dose in Milligrammes.	Amount of Blood Removed by Venesection in Cubic Centimetres.	Result.
53·6	1·25	6·7	280	Died in twenty-four hours.
36·2	1·25	4·5	240	Paralysed in twenty-four hours, died on the thirteenth day.
30	1·2	3·6	300	Paralysed in twenty-four hours and remained so for six days; recovered completely by the twelfth day.
30·8	1·0	3·1	350	Paralysed in twenty-four hours and remained so till the twelth day; recovered.

The sheep which received only one certainly lethal dose recovered from his paralysis, but had severe contractures of the forelimbs from faulty postures during his prolonged paralysis. It was therefore killed on the fourteenth day. The sheep which received one and a quarter certainly lethal doses and died on the thirteenth day, would probably have recovered with more careful nursing. The results are much less promising than those obtained after injection of the venoms of the death adder and tiger snake. In comparison with Fairley's ligature experiments (in which the venom was also injected in a concentration of 10 milligrammes per cubic centimetre) they indicate a definite though small advantage gained by the treatment. In Fairley's experiments sheep weighing 45·4 and 46·8 kilograms received one certainly lethal dose. One died in seventeen hours and the other recovered after being paralysed for two days. Sheep weighing 45·7, 45·9 and 45·8 kilograms received one and a quarter certainly lethal doses and all died within fifteen hours. Finally a sheep weighing 46·8 kilograms received one and a half certainly lethal doses and died in 57¾ hours.

The bites of the common brown snake, *Demansia textilis*, and of other species in this genus present a difficult problem in treatment. Despite the very poor biting apparatus of these snakes (Fairley) and the very small yield of venom obtained on milkings them, the bite has a not inconsiderable mortality (18·7%, Tidswell[11]). The snakes are are not easily collected in large numbers, and when caught rarely yield more than 6 milligrammes of venom at a milking, the average yield in captivity being extremely small. Tidswell[11] gives the average venom yield in thirteen observations on *Demansia textilis* as 4·8 milligrammes of dry venom, his maximal yield being 5·56 milligrammes. Fairley[12] obtained no venom from any of the seven living specimens he observed. My own figures for sixty-two milkings of thirteen brown snakes (*Demansia textilis*) show a maximal yield of 7·2 milligrammes and an average yield of 2·1 milligrammes. These considerations make it unlikely that sufficient venom from this species can be collected for the manufacture of univalent antivenine, though possibly sufficient may be obtained for use in the preparation of a polyvalent antivenine.

The small quantity of venom injected at a bite and the coagulant action of the venom, which contains a powerful thrombase, are circumstances favourable to treatment by the method of local venesection. I have therefore studied the certainly lethal dose of this venom by subcutaneous and by intravenous injection in sheep, the effects of treatment by simple ligature, and finally that by "local venesection."

The subcutaneous injection of doses not greatly in excess of the certainly lethal dose revealed an exceedingly interesting phenomenon. Most of the animals died rapidly with thrombosis in the heart, pulmonary veins, portal veins and inferior *vena cava*, the venom gaining entry into the veins in the neighbourhood of the injection. Great care was taken to avoid damage to small veins and the piston

of the syringe was always withdrawn before the injection was made so as to be sure that a vein had not been entered. In a few animals the injection was given intradermally with precisely the same result. The thrombase of this venom is very potent in the sheep and appears to be much more diffusible than that of other venoms. Possibly, however, the cytolytic constituents of the venom damage the walls of the small vessels at the site of the injection and so facilitate its entry into the bloodstream. In this respect the venom differs from that of the tiger snake and black snake with which I have not observed this phenomenon except with overwhelming doses in small animals (Martin[13]).

The results of subcutaneous and intradermal injection of the venom in sheep are set out in Table V. The venom was used in a concentration of 4 milligrammes per cubic centimetre.

TABLE V.

The Certainly Lethal Dose of the Venom of Demansia textilis by Subcutaneous or Intradermal Injection in Sheep.

Weight of Sheep in Kilograms.	Dosage in Milligrammes per Kilograms.	Total Dose in Milligrammes.	Method of Injection.	Result.
30·8	0·04	1·23	Subcutaneous	Died in forty-five minutes; thrombosis in heart, lungs, portal veins and inferior *vena cava*.
50·7	0·03	1·52	Subcutaneous	Died in twenty-five minutes, with thrombosis in the heart, pulmonary and portal veins.
39·0	0·03	1·17	Subcutaneous	Died in thirty-one minutes, with intravascular thrombosis.
30·0	0·02	0·6	Subcutaneous	Paralysed in twenty-six hours, died in fifty hours.
49·8	0·02	1·0	Intradermal	Died in eighteen minutes, with intravascular thrombosis.
39·0	0·02	0·8	Intradermal	Died in twenty-five minutes, with intravascular thrombosis.
48·0	0·01	0·48	Subcutaneous	Ataxia on the second day, weakness of hind limbs to fifth day; recovered.
41·5	0·01	0·41	Subcutaneous	Died in less than two hours, with intravascular thrombosis.

All but two of these animals died of intravascular thrombosis and the single survival with 0·01 milligramme per kilogram indicates that 0·02 cannot be greatly in excess of the subcutaneous certainly lethal dose. Only one animal of those which received this last dose, died with neurotoxic symptoms early on the third day, having escaped the early danger of intravascular thrombosis. In two there was thrombosis in the veins of the leg leading from the site of injection near the metacarpo-phalangeal articulation. The subcutaneous certainly lethal dose has been taken as 0·02 milligramme per kilogram, but it should be clearly understood that this is probably somewhat too high.

The symptoms in these animals were remarkably uniform. There was a period of uneasiness which terminated in sudden collapse and the animal remained "down" with occasional convulsive attacks which were of a striking nature. The head and neck were stretched out, the eyes rolled (in some vertically), then the body was stiffened and there were kicking or locomotory movements of the limbs. Death usually took place in a final convulsion. The corneal reflex was not lost till just before death.

Post mortem there was usually whipped out fibrin in both sides of the heart, with hæmorrhage in the endocardium. The pulmonary veins were clotted. The inferior *vena cava* was frequently partially clotted and the blood in the portal vein was solidly thrombosed. If the animal survived any length of time, the intestines became intensely engorged and hæmorrhagic, the spleen was enlarged and almost black on section. The liver was dark blue and much enlarged from the damming back of fluid blood which leaked out gradually after the cava had been divided, leaving the organ with a nearly normal colour though still intensely congested.

I next proceeded to ascertain the approximate certainly lethal dose of the venom by injection in a concentration of 0·2 milligramme per cubic centimetre into a vein behind one of the carpal joints. The results of these experiments are set out in Table VI.

TABLE VI.

Results of Intravenous Injection of the Venom of Demansia textilis into Sheep.

Weight in Kilograms.	Dose in Milligrammes per Kilogram.	Total Dose in Milligrammes.	Result.
48·9	0·005	0·24	Died in three and a half minutes.
34·5	0·002	0·069	Died in twelve minutes.
23·0	0·002	0·046	Died in three and a half minutes.
41·0	0·001	0·041	Died in nineteen minutes.
43·7	0·001	0·044	Died in five minutes.
40·5	0·001	0·041	No symptoms; survived.
33·0	0·001	0·033	Died in eleven minutes.
34·0	0·0005	0·017	No symptoms; survived.

The certainly lethal dose is probably not far removed from 0·001 milligramme per kilogram, the only animal surviving after injection of this dose having been previously injected subcutaneously with 0·002 milligramme of brown snake venom and having survived after treatment by local venesection.

Tidswell[11] gave the certainly lethal dose of this venom by subcutaneous injection in the rabbit as 0·2 milligramme per kilogram (the venom being of about the same order of toxicity for this species as that of the death adder and about a quarter as potent as that of the tiger snake). For the sheep, as judged by the results of subcutaneous injection, the venom is definitely more potent than that of the death adder, but only about half as potent as that of the tiger snake. By intravenous injection in sheep the venom is about five times as potent as that of the tiger snake. The subcutaneous-intravenous index for the venom is approximately 20 for the sheep as against an index of two for the venom of the tiger snake.

On these grounds it might be expected that ligature alone would be a highly effective procedure in the sheep. As will be seen, the results set out

in Table VII do not justify this hope. In three of these experiments the ligature was applied round the metacarpal to make certain that no arterial blood entered the part while the ligature was in position, and in one it was placed above the carpal joint as in the venesection experiments. In all cases it was applied for one hour. The venom was injected in a concentration of 4 milligrammes per cubic centimetre. Only one sheep survived, but in two the death time was greatly delayed and the animals were saved from the rapid coagulant action of the venom.

TABLE VII.

Results of Simple Ligature One Hour following the Injection of Brown Snake Venom in a Concentration of Four Milligrammes per Cubic Centimetre.

Weight in Kilograms.	Dose in Milligrammes per Kilogram.	Total Dose in Milligrammes.	Result.
Ligature applied round Metacarpal.			
43·2	0·02	0·86	Died in forty-eight minutes after removal of ligature; cardiac and portal thrombosis.
39·1	0·02	0·78	Very severe hæmorrhage from bowel, otherwise well after twenty-four hours; died in thirty-eight hours.
37·6	0·015	0·56[1]	Dyspnœa and uneasiness thirteen minutes after removal of ligature; in twenty-four hours, salivation, weakness of limbs; died on fourth day.
Ligature applied above Carpal Joint.			
38·0	0·02	0·76	Half an hour after removal of ligature, shaking, restless, dyspnœic; paralysed after thirty-six hours until the sixth day; recovery nearly complete thirteenth day.

[1] Intradermal injection.

Three of these animals exhibited neurotoxic symptoms and the results indicate that the certainly lethal dose by virtue of the neurotoxin of this venom is not far removed from that estimated on the basis of the thrombotic deaths which normally follow the subcutaneous or intradermal injection of brown snake venom in sheep.

In the two animals which died on the second and fourth days, the symptoms were salivation, weakness, ataxia and paralysis. Death occurred from respiratory failure. At autopsy the blood was fluid and there were extensive hæmorrhages in the lungs, thyreoid, thymus, pancreas and small intestine. The kidneys and adrenals were congested and the spleen was not enlarged. In the animal which died from portal thrombosis, the symptoms were those typical of intravascular coagulation. Slight dyspnœa and distress were present soon after removal of the ligature with sudden collapse and convulsions only two minutes before death.

The experiments in which local venesection was used for treatment are set out in Table VIII.

Protection against a single lethal dose was absolute, but against one and a quarter certainly lethal doses was not so complete and there was only delay in the death time after one and a half certainly lethal doses. The experiment on the second sheep which received 0·025 milligramme per kilogram was unsatisfactory. The venous ligature was insufficiently tight and enough venom entered the general circulation during the first period in which the arterial ligature was removed, to cause extensive intravascular thrombosis and death. In the other experiment in which this dose was given, the ligatures were left in position for some time before commencing treatment by venesection.

TABLE VIII.

The Results of Treatment by "Local Venesection" following the Injection of Brown Snake Venom.

Weight in Kilograms.	Dose in Milligrammes per Kilogram.	Total Dose in Milligrammes.	Amount of Blood Removed by Venesection in Cubic Centimetres.	Result.
38·5	0·03	1·15	180	Died on the sixth day.
41·0	0·025	1·02	360	No symptoms of envenomation, but was destroyed on the seventh day owing to severe local damage to the limb.
40·8	0·025	1·02	140	Died from intravascular thrombosis twenty-two minutes after the injection.
41·6	0·02	0·84	320	Survived without symptoms.
36·7	0·02	0·73	350	Survived without symptoms.

The obvious difficulty in applying this treatment is the practical one that snake bites seldom occur when a doctor is at hand to treat the snake-bitten person. After an "arterial" ligature has been at once placed in position and the ordinary measures of local treatment have been applied, some time may elapse before a doctor can be brought to the patient or the patient taken to the doctor. If this interval be half an hour and not more the ligature may safely be left in position, without loosening it to permit blood to flow into the limb and to re-establish the circulation momentarily, since in this country we are not concerned with the disastrous local effects met with, even without prolonged ligature, after bites by vipers. If the interval be longer, occasional loosening of the ligature, say every twenty minutes after the first half hour, may be necessary to avoid the risk of permanent damage to the limb from the prolonged anoxæmia of the tissues. Of course every time this is done without venesection being performed, some venom will enter the circulation and the value of treatment by venesection when finally carried out will be less. Some experiments in which "arterial" and "venous" ligatures above the carpal joint have been left on for an hour or more after the injection of venom, before commencing the treatment by venesection, were directed towards ascertaining whether under these conditions venom would be removed from the bitten part in sufficient quantity to make the treatment worth while. In these experiments the scales were weighted against success by the fact that during the course of the

first hour there was probably a small but steady leak of blood from the limb into the circulation since the ligature was placed above the carpal joint. Only three of these experiments were made, one with brown snake venom, one with tiger snake venom and a third with the venom of the death adder. The "arterial" ligature was not applied round the metacarpal bone because such a small part of the limb would be ligated off and the conditions would not be in any sense comparable with those applying in ligature round the arm or thigh in man.

The results of these experiments were not so striking as those reported earlier in this paper, but, even under these circumstances, the procedure appeared to be of value after the injection of coagulant venoms.

The experiments were relatively unsuccessful, not on account of fixation of venom in the tissues, but because the "arterial" ligature applied above the carpal joint in sheep is not perfectly efficient. In a single experiment with the venom of the death adder the treatment was without avail.

A sheep weighing 41·4 kilograms had the two ligatures applied above the carpal joint of the left forelimb at 10.53 a.m. and 1·55 milligrammes (about one and a half certainly lethal doses) of death adder venom were injected subcutaneously in a concentration of 4 milligrammes per cubic centimetre. At 1.40 p.m., that is two hours forty-seven minutes later, treatment by venesection was instituted until 2.50 p.m., when the ligatures were finally removed. Bleedings were obtained at 1.40, 1.47, 1.54, 2.0, 2.5, 2.10, 2.20, 2.35, 2.45 and 2.50 p.m. to a total volume of 360 cubic centimetres. The animal presented no symptoms up to 10 p.m., but was found dead at 8 a.m. the following morning.

In this experiment the "arterial" ligature was on uninterruptedly for two hours and forty-seven minutes, and intermittently for an additional period of one hour and ten minutes. The ligated limb was not swollen, so that if any inflow of arterial blood took place, a corresponding outflow of venous blood must have occurred. Probably ample time was allowed for the absorption of a lethal dose before treatment by venesection was begun.

Another experiment was carried out on June 5, 1930, with tiger snake venom.

The sheep weighed 34 kilograms and after the application of the two ligatures above the carpal articulation one and a half certainly lethal doses of venom (0·51 milligramme) were injected subcutaneously in the usual site at 10.25 a.m. Bleedings were made at 11.50 and 11.54 a.m., 12.1, 12.9, 12.15, 12.18, 12.21, 12.29, 12.33, 12.38 and 2.45 p.m. when the ligatures were finally removed. During the period between the bleedings when the arterial ligature was applied there was a steady though small leak of blood. Evidently at no time was the "arterial" ligature thoroughly efficient. In all 320 cubic centimetres of blood were removed by venesection. The animal presented no symptoms until 10 a.m. on June 7, 1930, when it went "down" with paresis of the limbs. It remained paralysed for eight days and then developed diarrhœa which rendered its condition so miserable that it was killed on the thirteenth day.

In this experiment the "arterial" ligature was applied uninterruptedly for one hour twenty-five minutes and intermittently for an additional period of fifty-five minutes. Though applied as tightly as possible it was unusually inefficient. Despite this a good deal of venom must have been removed by the treatment.

In the third experiment with brown snake venom 0·025 milligramme per kilogram (one and a quarter certainly lethal doses) was injected and the ligatures were left in position for fifty minutes before the commencement of treatment by venesection. During the following eighty minutes before the final removal of the ligatures 360 cubic centimetres of blood were removed in ten bleedings. The animal presented no symptoms, but developed sepsis in the leg on the third day which showed no sign of abatement by the sixth day. It was therefore destroyed.

This was the only example of severe sepsis met with in these experiments, though in the tiger snake experiments, in which also the vein used for venesection had to be tied, there was in several some local suppuration since it was not possible to keep the wounds perfectly clean in animals which were "down," despite daily dressing of the wounds and frequent cleaning out of the stable in which the sheep were housed. Martin recommended that ligature in man should not be left on longer than an hour altogether for fear of causing damage to the ligated part. In sheep, ligatures placed round the metacarpal bone have been left on two and three hours without permanent untoward effects when no venom has been injected. It is perhaps significant that in the experiment with brown snake venom just recorded, the severe sepsis which occurred followed rather prolonged ligature, though in the preceding experiment with tiger snake venom the ligature which was somewhat inefficient, was left on even longer without any serious local damage resulting.

Fairley[5] has summarized the death times of eighty-one fatal cases of snake bite in Australia. Nearly 40% of these occurred in twenty-four hours or more, and it is highly probable that in these not more than one and a half certainly lethal doses were absorbed. It is in this group of cases that local venesection, if applied early enough, may be expected to be effective. The vein opened should be one draining as directly as possible the region where the bite is situated, though no attention has been paid to this point in the foregoing experiments.

In cases where a much larger dose has been given, the method is worthy of trial though my experiments indicate that it will not remove more than one-third or one-half of the venom injected even if applied immediately. Such a decrease in the amount of venom will diminish the quantity of antivenine necessary, and where it is doubtful whether the amount of antivenine available is adequate for treatment this method should certainly be tried. It is advisable to have the area where the "arterial" ligature is applied well covered with clothes to avoid damage to the tissues by the repeated application of the ligature.

There can be no question that the treatment of the bite should consist of excision of an area round the fang punctures $3·2 \times 1·9$ centimetres immediately after application of the ligature. In Fairley's experiments on the value of this treatment in sheep following natural bite by the tiger snake, protection must have been afforded against several lethal doses of venom. If such treatment as amputation of a finger or toe or immediate excision of the bitten area is to be effective, it must be applied at once, and since a medical man is hardly ever present at the time of the bite,

this treatment must be left to the layman in most cases.

It may be argued that it is dangerous to advise lay persons to carry out such procedures, but if the area bitten be upon a limb, the ligature will control any hæmorrhage until medical help can be obtained and the amount of bleeding, when the ligature is lifted to obviate permanent damage to the ligated part, need not be excessive if only small arteries are involved. Local venesection will probably be of use even when it is not carried out immediately provided that an efficient ligature has been applied directly after the bite and left in position as long as is practicable.

Summary and Conclusions.

1. Experiments upon sheep injected with the venoms of the death adder and tiger snake have demonstrated the value of treatment by local venesection when this can be applied immediately. Those with copper-head and brown snake venoms were less satisfactory.

2. Protection was afforded against from one to one and a half certainly lethal doses of the venoms of the death adder and tiger snake and against from one to one and a quarter certainly lethal doses of the venoms of the brown snake and copper-head.

3. The ligatures in these experiments were applied above the carpal joint in the forelimb and therefore the blood flow to the ligated part, though very nearly, was not always completely obstructed. This did not seriously affect the results when venesection was carried out at once, but where treatment was delayed to make the experiments more comparable with practical conditions, the results were not so satisfactory.

4. The certainly lethal doses of brown snake venom by subcutaneous and intravenous injection have been determined for sheep. The "thrombase" of this venom is extremely potent in this species, the subcutaneous-intravenous index being approximately 20.

5. Despite this high index, treatment by simple ligature round the metacarpal bone was not effective.

6. The "thrombase" of brown snake venom is apparently highly diffusible and, when injected subcutaneously or even intradermally, finds its way rapidly into the venous blood in sufficient concentration to cause death by intravascular coagulation.

7. In bites by the brown snake it is urgently necessary to apply the ligature immediately and excise the bitten area. Treatment by venesection, as soon as this is practicable, should prove of definite value in cases where excision has not been done. It is unlikely that univalent antisera will ever be available for treatment of the bites of this snake or of other species of the genus *Demansia*.

8. These experiments afford further evidence of the absorption of Australian snake venoms directly into the bloodstream.

Acknowledgements.

The experiments were carried out at the Veterinary Research Institute and my thanks are due to Dr. Albiston and his staff and to Mr. A. W. Turner and Mr. Murnane of the research staff of the Council of Scientific and Industrial Research for their kindly cooperation and interest in this work.

References.

(1) N. Hamilton Fairley: "Criteria for Determining the Efficacy of Ligature in Snake Bite," THE MEDICAL JOURNAL OF AUSTRALIA, March 23, 1929, page 377.
(2) C. J. Martin: Allbutt's "System of Medicine," 1897, Volume II, page 828.
(3) C. J. Martin and G. Lamb: Allbutt and Rolleston's "System of Medicine," 1907, Volume II, Part II, page 783.
(4) H. W. Acton and R. Knowles: "Studies in the Treatment of Snake Bite," *Indian Journal of Medical Research*, 1914, Volume II, page 91.
(5) N. Hamilton Fairley: "The Present Position of Snake Bite and the Snake-bitten in Australia," THE MEDICAL JOURNAL OF AUSTRALIA," March 9, 1929, page 310.
(6) C. J. Martin: "On the Physiological Action of the Venom of the Australian Black Snake (*Pseudechis porphyriacus*)," *Journal of the Royal Society of New South Wales*, 1895, Volume XXIX, page 147.
(7) C. H. Kellaway: "Local Venesection in the Treatment of Snake Bite," THE MEDICAL JOURNAL OF AUSTRALIA, April 26, 1930, page 551.
(8) C. H. Kellaway: "The Venom of *Notechis scutatus*," THE MEDICAL JOURNAL OF AUSTRALIA, March 16, 1929, page 353.
(9) C. H. Kellaway: "A Preliminary Note on the Venom of the Australian Copper-head (*Denisonia superba*): Its Toxic Effects in the Common Laboratory Animals," THE MEDICAL JOURNAL OF AUSTRALIA, March 16, 1929, page 361.
(10) C. H. Kellaway: "Observations on the Certainly Lethal Dose of the Venom of the Death Adder (*Acanthophis antarcticus*) for the Common Laboratory Animals," THE MEDICAL JOURNAL OF AUSTRALIA, June 8, 1929, page 768.
(11) F. Tidswell: "Researches on Australian Snake Venoms," 1906.
(12) N. Hamilton Fairley and Beryl Splatt: "Venom Yields in Australian Poisonous Snakes," THE MEDICAL JOURNAL OF AUSTRALIA, March 16, 1929, page 347.
(13) C. J. Martin: "Observations upon Fibrin Ferments in the Venoms of Snakes and the Time Relations of Their Action," *Journal of Physiology*, 1905, Volume XXXII, page 207. "On the Physiological Action of the Venom of the Black Snake (*Pseudechis porphyriacus*)," *Ibidem*, 1895, Volume XXIX, page 192.

THE INFLUENCE OF EXTRACTS OF PRICKLY PEAR ON DIABETES MELLITUS.[1]

By W. WILSON INGRAM, M.C., M.D. (Aberdeen),
Honorary Physician, Royal North Shore Hospital of Sydney;

AND

G. V. RUDD, M.Sc. (Melbourne),
Biochemist, Royal North Shore Hospital of Sydney.

VARIOUS substances have been suggested from time to time as adjuncts to "Insulin" therapy. These may be divided into three groups, namely: (i) Substances produced synthetically, (ii) substances elaborated from animal sources, (iii) substances prepared from plants. It is with the third group only that we are here concerned.

Mark and Wagner(1) and Eppinger, Mark and Wagner(2) found that an infusion of blueberry leaves had a beneficial effect on glycosuria. They separated in a more concentrated but still impure form

[1] This work was sustained in part by the T. E. Rofe Foundation.

an active principle which they named "myrtillin" and which had a controlling action on glycosuria and hyperglycæmia. Further inquiry was pursued by Allen,[3] who was thus led to believe "that 'myrtillin' plays some accessory part in carbohydrate metabolism and that if properly used it will prove valuable as an accessory factor in diabetic treatment." He stated also that: "Myrtillin occurs in all green plants." His work was corroborated by Shpiner.[4] Mills[5] has reported the successful treatment of *diabetes mellitus* with extracts of plants rich in vitamin *B*. These were prepared by extracting the plants with a 0·4% solution of hydrochloric acid in 40% alcohol.

In this country two plants have been stated to be of value in the treatment of *diabetes mellitus*. They are *Vinca rosea* and *Opuntia enermis* (prickly pear). With regard to *Vinca rosea*, Nye and Fitzgerald,[6] Lee and Drew[7] and Corkill and Doutch[8] have been unable to demonstrate any definite effect on the blood sugar or urinary sugar of extracts or infusions of the leaves. In their experiments both patients and animals were used and in no case was there obtained a result which encouraged the belief that extracts of this plant would be of value as a therapeutic measure in *diabetes mellitus*. With regard to prickly pear, Shellshear[9][10] has administered an extract of this plant to diabetic patients and has claimed favourable results. In view of these conclusions and the belief held popularly in this country that an extract of prickly pear has some efficacy in the treatment of *diabetes mellitus*, investigation of the question was undertaken.

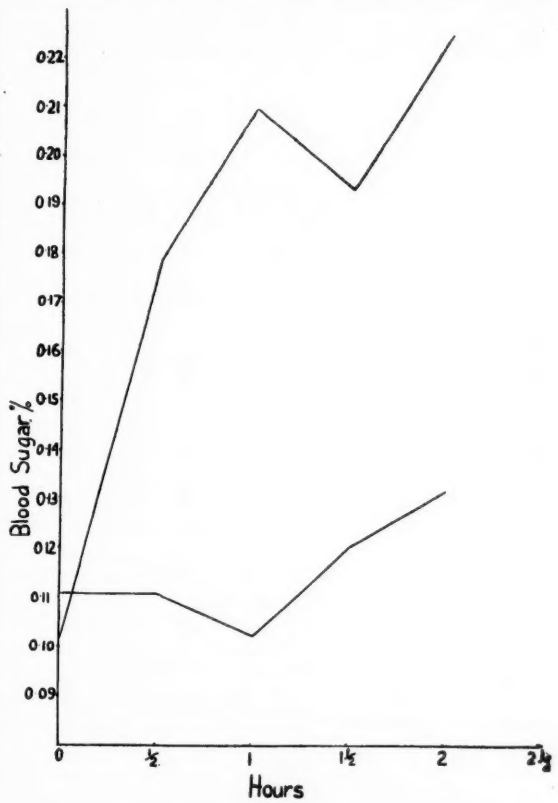
First, an attempt was made to discover any influence of an extract of prickly pear on the response of a normal rabbit to a glucose tolerance test. For this purpose, a dose of nine cubic centimetres of glucose solution containing three grammes of glucose was given to an animal which had been starved for eighteen to twenty-four hours, blood sugar estimations being done before and every half hour after the administration of the glucose. A stomach tube improvised from a number four rubber catheter and passed into the rabbit's stomach through a hole in a wooden mouth gag, was used for the introduction of the glucose solution. It was found, however, that animals starved for eighteen to twenty-four hours showed very divergent results of the test. This was attributed to the fact that the stomachs of the animals contained varying quantities of food materials which exerted an adverse influence on both the rate and constancy of absorption of glucose into the blood stream. Thus the glucose tolerance curves varied between a line showing a small increase in blood sugar above normal and a line showing a rapid and maintained increase to a blood sugar of approximately 0·20%.

Nevertheless, a second series of tests was carried out in which various extracts of prickly pear were added to the glucose solution on administration, but no change in the range of results was noted. Graphs I and II show the results obtained in the tests. It was apparent, therefore that this method of investigation was incapable of yielding useful results. It failed to demonstrate the presence of any very active blood sugar lowering principle in the following extracts:

1. Various fractions of the strained extract of equal weights of minced prickly pear and water, saturated with sodium bicarbonate.

2. The strained extract of equal weights of minced prickly pear and water, stirred and allowed to stand overnight.

3. The strained extract of equal weights of minced prickly pear and water, boiled and allowed to cool.

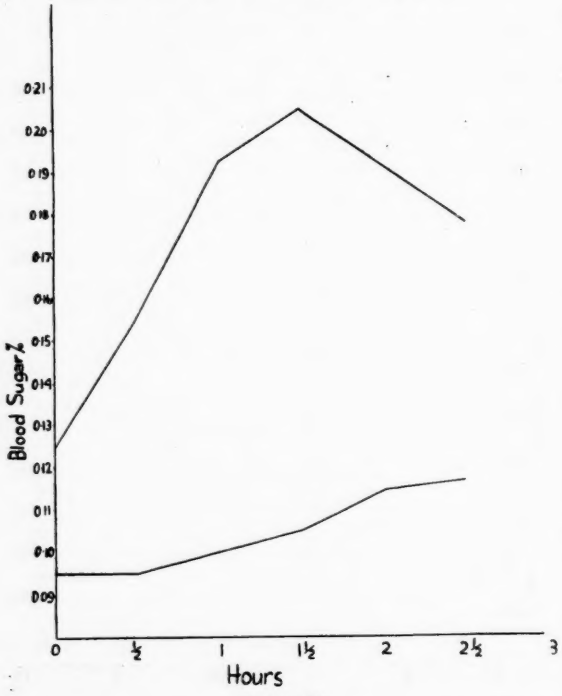


GRAPH I.

Showing extremes of results found in glucose tolerance tests on starved rabbits, after the administration of three grammes of glucose.

A new line of investigation was then followed, myrtillin being prepared in some quantity by a method which followed the outline of the procedure as given by Allen.[1] This was done with the object of observing the effects of the preparation administered orally to diabetic patients. Briefly the procedure adopted for the preparation of myrtillin from prickly pear was the following. The well minced prickly pear was mixed with such a quantity of alcohol as would make the alcohol content of the extract 55% by volume, hydrochloric acid being added during the extraction so that the mixture

would contain 0·3%. After standing overnight, the mixture was strained and the residue of prickly pear was reextracted with 55% alcohol. The mixture was strained and the two extracts combined. Clarification of the extract was effected by filtration through cotton wool and then through paper pulp. The alcohol content of the solution was determined and sufficient rectified spirit added to bring the alcohol concentration to 75% volume, the acidity being largely neutralized by the addition of 10% sodium hydroxide. The precipitate was filtered off and a small quantity of saturated ammonium sulphate solution was added with constant vigorous shaking to the filtrate. The precipitate which was filtered off, corresponds to Allen's myrtillin and was obtained as a white powder in a yield of one gramme from 500 grammes of prickly pear.



GRAPH II.

Showing extremes of results found in glucose tolerance tests on starved rabbits, after the administration of three grammes of glucose and various extracts of prickly pear.

The following are the results of administration to diabetic patients of 0·5 gramme of the powder (the equivalent of 250 grammes of prickly pear) twice daily half an hour before the midday and evening meals.

CASE I.—A male, *ætatis* fourteen years, was admitted to hospital from the diabetic clinic. He was being given twenty-eight units of "Insulin" daily with a diet of 1,700 calories which was adhered to throughout his stay in hospital. His blood sugars varied between 0·070% and 0·077%, so that he had a tendency to hypoglycæmia. He was given 0·5 gramme of prickly pear extract twice daily and his "Insulin" was reduced to twenty-six units daily. His blood sugar then rose to 0·094%. His "Insulin" was reduced by successive decreases of two units during the next fourteen days to twenty-two units per day. His blood sugar rose to 0·19% and sugar appeared in the urine. He was kept on this treatment for the next twenty days, in which period his blood sugar varied between 0·14% and 0·20% and glycosuria appeared intermittently. During the next sixteen days the administration of the prickly pear extract was stopped and he was kept on the same dose of "Insulin" and the same diet. In this period the blood sugar varied from 0·14% to 0·18% and glycosuria appeared occasionally. For the next twenty-five days the extract was again administered with "Insulin" dosage and diet as before. His blood sugar varied from 0·13% to 0·20% and intermittent glycosuria was noted. The prickly pear extract was now stopped and his "Insulin" dosage increased to twenty-six units daily. During the next twenty-five days his blood sugar varied from 0·11% to 0·15% and the glycosuria disappeared.

It was quite evident in this case that the prickly pear extract had no effect on the blood sugars or on the glycosuria, whereas an increase in "Insulin" dosage of four units per day was sufficient to keep his blood sugar at a lower level and to abolish the glycosuria.

CASE II.—A male, *ætatis* forty-six years, attending the diabetic clinic at the hospital, was admitted as an in-patient. He was receiving thirty-eight units of "Insulin" daily and a fixed diet, but had been careless in dieting and had marked glycosuria. Within seven days his blood sugar had fallen to 0·13% and his glycosuria had disappeared. On the ninth day the administration of prickly pear extract was commenced. The "Insulin" dosage was reduced to thirty-six units and then to thirty-two units, upon which the blood sugar rose to 0·20% and glycosuria reappeared. A further reduction to twenty-eight units led to continuous glycosuria and blood sugar of 0·20%, which were not improved by an increase in the "Insulin" dosage to thirty units.

In this case the hyperglycæmia and glycosuria resulting from an "Insulin" reduction of six units were not influenced by the administration of the prickly pear extract.

CASE III.—A female, *ætatis* twenty-three years, attending the diabetic clinic, was admitted to hospital, receiving twenty-two units of "Insulin" daily with a fixed diet. Her blood sugar was 0·08% and she had no glycosuria. She was given prickly pear extract for the next twenty-two days and her "Insulin" dosage was gradually reduced to fourteen units daily, on which dose her blood sugars varied from 0·11% to 0·17% without sugar appearing in the urine. The prickly pear extract was now stopped and the patient was transferred to the diabetic clinic. She was given fourteen units of "Insulin" daily and her blood sugars varied from 0·08% to 0·11%, the urine remaining sugar free.

In this case it appears that careful dietary control was responsible for the reduction of "Insulin" dosage as no increase in blood sugar followed a ten-day period in which no extract was given.

CASE IV.—A female, *ætatis* thirty-eight years, was admitted to hospital from the diabetic clinic, receiving twenty-four units of "Insulin" daily on a fixed diet. She showed no glycosuria and her blood sugar was 0·10%. Administration of prickly pear extract was commenced and continued for twenty-five days. Her "Insulin" dosage was gradually reduced over a period of twenty-one days to eighteen units daily, when her blood sugar rose to 0·18% and glycosuria reappeared. The prickly pear extract was then stopped. She was kept under observation in the diabetic clinic for several months, during which period her blood sugars usually varied between 0·12% and 0·15% and she showed no glycosuria.

The reduction of "Insulin" dosage by six units in this case was apparently due to better dietary

control, as the patient remained sugar free and her blood sugar remained near normal limits for several months on eighteen units of "Insulin" daily without the administration of the extract.

CASE V.—A male, *ætatis* forty-eight years, was admitted to hospital with glycosuria, ketonuria and hyperglycæmia. He was given a fixed diet and finally his urine became sugar free and his blood sugar was 0·11%, with his "Insulin" dosage of twenty-two units daily. Administration of the prickly pear extract was commenced. His "Insulin" was reduced two units at a time, to twelve units daily on the twenty-second day. His blood sugar rose to 0·21% and glycosuria reappeared. The prickly pear extract was then stopped. He was kept under observation for twelve months, in which period his blood sugar did not rise above 0·14% and he showed no glycosuria.

The prickly pear extract had no apparent effect in the treatment of the patient, as he was able to maintain himself in a normal condition on the reduced dose of "Insulin" without taking the extract.

This series of five cases failed to show that the extract had any effect on the urinary or blood sugar. The reductions that were possible in the "Insulin" dose may be explained by more careful control of the patients' diets in hospital. The fact that the patients, when being discharged, were very definitely instructed to exercise the utmost care in preparing their food, would make the patients' own dietary control almost equal to that of the hospital. In these circumstances, then, they remained without glycosuria and hyperglycæmia on their reduced "Insulin" dose. The suggestion that some of the cases receiving the extract were permanently improved is invalidated by Case I in which prolonged administration of the extract had to be followed by a change of "Insulin" dosage to the original level in order that the hyperglycæmia and glycosuria might be cleared up. The patient in Case III also was readmitted to hospital some months later so much worse that her "Insulin" dosage had to be increased to sixty units daily.

Macleod,[11] in a recent discussion of "Insulin" substitutes, said that the laboratory test for a proposed "Insulin" substitute was its ability to remove the symptoms of a diabetic animal. He had received for testing no proposed substitutes that could fulfil this condition. In his view, "Synthalin" did so only very slightly and had no action at all comparable with that of "Insulin." The same considerations applied to myrtillin.

The present work is seen definitely to indicate that myrtillin prepared from prickly pear does not satisfy Macleod's criterion for an "Insulin" substitute.

Not only was it found impossible to demonstrate any effect on the glucose tolerance curves of normal rabbits by various extracts of prickly pear, but also a myrtillin-like substance prepared from prickly pear had no demonstrable effect when administered to diabetic patients.

References.

[1] R. E. Mark and R. I. Wagner: Quoted by Allen, *loco citato.*

[2] H. Eppinger, R. E. Mark and R. I. Wagner: Quoted by Allen, *loco citato.*

[3] F. M. Allen: "Blueberry Leaf Extract," *The Journal of the American Medical Association*, November 5, 1927, page 1577.

[4] L. B. Shpiner: "The Action of Myrtillin in Pancreatic Insufficiency," *American Journal of Physiology*, 1928, Volume LXXXIV, page 396.

[5] C. A. Mills: "Treatment of Diabetes with an Acid Alcoholic Extract of Plants Rich in Vitamin B," *The American Journal of the Medical Sciences*, March, 1928, page 376.

[6] L. J. J. Nye and Margaret E. Fitzgerald: "Vinca Treatment of Diabetes," THE MEDICAL JOURNAL OF AUSTRALIA, November 17, 1928, page 626.

[7] D. H. K. Lee and W. R. M. Drew: "Vinca Rosea: Its Effect on the Blood Sugar Level of Normal Rabbits," THE MEDICAL JOURNAL OF AUSTRALIA, May 25, 1929, page 699.

[8] A. B. Corkill and A. Doutch: "An Investigation into the Alleged Therapeutic Properties of Vinca Rosea in the Treatment of Diabetes," THE MEDICAL JOURNAL OF AUSTRALIA, February 15, 1930, page 213.

[9] W. G. Shellshear: "The Treatment of Diabetes by Insulin and Prickly Pear," THE MEDICAL JOURNAL OF AUSTRALIA, March 28, 1925, page 329.

[10] W. G. Shellshear: "The Prickly Pear and Diabetes," THE MEDICAL JOURNAL OF AUSTRALIA, February 6, 1926, page 175.

[11] J. J. R. Macleod: "Diabetes as a Physiological Problem," *The British Medical Journal*, May 17, 1930, page 918.

THE WHITE CEDAR OR AZEDARACH.

By JOHN MACPHERSON, M.A., B.Sc., M.B., Ch.M.,
Lecturer in Therapeutics and Materia Medica, University of Sydney.

IN the Indian and Colonial Addendum (1900) to the British Pharmacopœia (1898) was included the dry bark of *Azadirachta Indica* (Indian Azadirach) belonging to the family *Meliaceæ*. This was the bark of the stem of the Neem (or Nim) or Margosa tree (*Melia Azadirachta*). Other names for the tree are Pride of India, Pride of China, Azedarach, bead tree, Cape lilac (erroneously), Australian lilac, but in Australia it is generally known as white cedar. Linnæus in 1753 first described the tree under the name *Melia Azedarach*, the Australian tree being given the variety name later of *australasica*. The *Melia composita* of Willdenow and *Melia australis* are but synonyms. The Azedarach extends from China and India through the east Indies to the northern and eastern coastal areas of Australia, including Queensland and New South Wales. It has been grown in Southern Europe and the Mediterranean. Greek monks planted it about their monasteries and used the strongly ribbed seeds as beads; hence the name "bead tree." It has been introduced also to Brazil and is now naturalized in the Gulf States of the United States of America. It grows as well in Norfolk Island, but whether indigenous or introduced is not quite certain. The white cedar is one of our most beautiful trees, attaining a height of forty feet, and is one of the few deciduous trees indigenous to Australia. The lilac-coloured inflorescence in massed panicles is particularly beautiful, giving off a delightful frag-

rance, especially at night. The tree yields a gum resembling that of the wattle (*Acacia*), the cherry and the plum.

The Hindus employ the bark, leaves, flowers and fruit for many medicinal purposes. The bark is almost inodorous. Its taste is at first sweetish, but afterwards bitter and somewhat nauseating. The "Addendum" contained two preparations: The infusion (one in 100)—dose, a half to one fluid ounce (fifteen to thirty mils)—and the tincture (one in ten)—dose, a half to one fluid drachm (two to four mils). In the United States of America the fresh root bark is mostly employed. Unofficial preparations are a decoction of a strength of two in thirty (boiled down to two in twenty). Of this preparation one half to one fluid ounce (fifteen to thirty mils) is given to a child every two or three hours, until the bowels are opened or vomiting results. A purgative may be given also if necessary. This treatment is as an anthelmintic. The other unofficial United States preparation is an alcoholic fluid extract with white sugar. A single dose of four mils (one fluid drachm) is administered. This preparation is said not to be so good as the decoction. The active principle has not been definitely established. Various bitter substances, including margosin, have been isolated, as well as a resin and a volatile oil.

The bark is slightly astringent. It is largely used as a bitter tonic (like quassia) in convalescence from fevers and other conditions. It is also reputed to be a febrifuge and antiperiodic in the milder types of malarial fever. Its main indication, however, is as an anthelmintic for round worms (*Ascaris lumbricoides*) and "bot" infestation in horses. The fresh root bark is the most active. Toxic symptoms may follow large doses, resembling poisoning by belladonna and spigelia (pink root). Poisonous properties have been known from the times of Arabian medicine, but some authorities are sceptical as to these occurrences. The root bark would apparently be the chief offender. The toxic manifestations recorded have been: Faintness, giddiness, dilated pupils, dimness of vision, mental confusion, stupor, stertorous breathing, cold sweats, vomiting and purging. These effects, however, pass off without any permanent injury to the system.

Particular interest attaches to the fruit, popularly termed a "berry," but really a drupe. The drupe is globular, yellowish-brown in colour and about the size of a rather small cherry. The question at issue is whether the fruit is or is not poisonous. In the United States it is not considered so. Both in the National Dispensatory and in Hare's "Practical Therapeutics" we have the definite assertion that the "berries" have very little toxic power and that children may eat largely of them without ill effects. In Australia they are looked upon with great suspicion as far as human beings are concerned. Dr. George Bennett was one of the keenest natural history observers in Australia many years ago. In his "Gatherings of a Naturalist," 1860, page 327, he states that the white pulpy exterior of the seeds is highly poisonous. He adds that Avicenna, the Arabian physician, cautioned people even against the leaves and wood. Hence the Arabs called the tree *Zederact* which signified poison. The seeds were never eaten. On the other hand Webster's Dictionary states that the name is derived from the Persian word *Āzāddirakht*, meaning a noble tree. The late Mr. J. H. Maiden, Government Botanist of New South Wales, recorded an instance of sudden and serious illness in an Adelong school pupil following the ingestion of the fruit. In regard to stock, including pigs, the "berries" are reputedly poisonous. Mr. H. Finnemore, at the Sydney Medical School, is at present undertaking investigations to clear the matter up and any information on this subject would be of the greatest value. The National Dispensatory states that the "berries" are attractive and nutritious food for horses. When we turn to the birds, however, we find that frugivorous species do eat them with impunity. Such birds are fruit pigeons, currawongs (black magpies) and others. But a rosella parrot of my own died suddenly after partaking of such food. It has been suggested that, as far as birds are concerned the poisonous constituent resides inside the seed or kernel which is extremely hard and in most birds passes through the intestine intact. In the case of birds with powerful beaks, such as cockatoos, such kernels would be broken and the toxic bodies liberated. A fixed oil has been expressed from the fruit, having a strongly bitter taste. It has been used externally as an embrocation for rheumatism and as an insecticide and internally as an anthelmintic.

The leaves have been employed as a local stimulant for indolent ulcers. It has been stated that cattle and horses are not injured by eating the leaves which are actually beneficial in "bot" infestation in horses.

THE TREATMENT OF BLINDNESS ASSOCIATED WITH RETINITIS PIGMENTOSA: A PRELIMINARY NOTE.

By N. D. ROYLE, M.D., Ch.M. (Sydney), F.C.S.A.,
Sydney.

RETINITIS PIGMENTOSA is a disease characterized by degeneration of the retinal tissues and an accumulation of pigment in the substance of the retina consequent upon degeneration and constriction of the retinal vessels. The idea that the disease is due to degeneration of retinal vessels is supported by experimental evidence. When the short posterior ciliary arteries are sectioned in the rabbit, there follows pigmentary degeneration of the retina similar to that seen in *retinitis pigmentosa*.[1] Another suggested cause is that the disease is due to a primary degeneration of nerve tissue, but it is doubtful that this occurs, apart from some alteration in the blood supply.

Subjective Signs of Disease.

The subjective signs of the disease are: Night blindness, loss of acuity of vision, contraction of fields and in advanced cases total blindness.

Treatment.

My attention was first directed to the pathological changes in the blood vessels in this disease by Dr. Lawrence, of Melbourne, and I thought that as division of the sympathetic supply of the eye dilates the vessels, the conditions underlying the disease might be altered with beneficial effect. Dr. E. A. Brearley, of Sydney, sent me the patients.

The Operation.

The operation I carried out upon these patients was thoracic sympathetic trunk section, and consisted in dividing the sympathetic trunk just about the level of the second thoracic ganglion. This operation has been described in THE MEDICAL JOURNAL OF AUSTRALIA.[2]

Results of Operation.

The first patient underwent operation on May 29, 1930. For the first week after operation he did not improve, but since then there has been steady improvement in the acuity of vision and in the enlargement of the fields of vision.

The patient had had *retinitis pigmentosa* for the past ten years. Prior to operation he could not see clearly; everything was misty; for example, he could not see fingers moving at a distance of 120 centimetres (four feet). He was totally blind in the dark. His central vision was absent, but he could distinguish lights of motor cars at night. He had suffered from blepharospasm of the right eye for three months prior to operation. Two weeks after operation he could tell the time on a clock with a two-inch dial, black figures on a white ground. He could also distinguish fingers moving at a distance of 120 centimetres (four feet). On June 18 he was very much improved; he could read letters 1·25 centimetres (half an inch) high and the face of an alarm clock with a ten centimetre (four inch) dial at a distance of 240 centimetres (eight feet). On June 24 he reported that the fields of vision were enlarged and that the vision of the left eye was also improved, although the right side was subjected to operation. He also reported that he could read small print in a good light. On July 4, 1930, he was able to read typewritten letters.

Dr. E. A. Brearley's report on the patient is to the effect that he was forty-three years of age and had first been seen on May 16, 1930. At this time the vision of the right and left eyes was less than $^6/_{60}$. There was insufficient fixation for testing the fields of vision. Definite *retinitis pigmentosa* was diagnosed. On June 18, 1930, the patient reported fuller fields of vision.

On June 30, 1930, the acuity of vision of the left eye was $^6/_{60}$ with correction. On July 14, 1930, the acuity of vision of the right eye was $^6/_{60}$ and of the left eye $^6/_{36}$.[1]

The other four patients have also had some improvement in the acuity of vision and enlargement of the fields of vision, but it is too early to state the results accurately.

References.

(1) J. Parsons: "Pathology of the Eye," Volume IV, 1908.
(2) N. D. Royle: "Sympathetic Trunk Section: A New Operation for Raynaud's Disease and Spastic Paralysis of the Upper Limb," THE MEDICAL JOURNAL OF AUSTRALIA, October 6, 1928.

[1] At the time of going to press a note has been received to the effect that the patient's field has increased 10% since July, being now three times the original field area.

Reports of Cases.

CEREBRO-SPINAL FEVER.

By W. E. HASKER, M.B., B.S.,
Resident Surgeon, Toowoomba General Hospital, Queensland.

THE clinical features and course run by a case of cerebro-spinal fever treated at this hospital brought out some few points which make it of sufficient interest to bring under the notice of the readers of the journal and to invite discussion on these manifestations.

The patient lived in the centre of the town and was engaged as a messenger boy; no other case of this nature was brought under notice of the authorities. The last case of cerebro-spinal fever entered in the hospital records occurred in 1924.

In the clinical investigation and treatment of the case invaluable and prompt help was given by the Commonwealth Health Laboratory.

The patient, D.C., aged fifteen years, was admitted to the isolation block at the Toowoomba General Hospital at 7 p.m. on May 6, 1930. He had been ill for four days, beginning on May 2, 1930. On Friday, May 2, 1930, he seemed to have a cold in the head. On May 3, 1930, at noon, he complained of frontal headache, had no appetite and his headache was getting worse. On May 4, 1930, he was semidelirious in the morning and crying out with headache; he was quite delirious in the evening. On May 5, 1930, all the symptoms were worse. Head retraction was also present; it became more pronounced and a spastic paralysis of the right arm and hand was present. This remained for four days. He had incontinence of urine. There was no vomiting and the bowels were opened with an enema. Lumbar puncture yielded fluid under increased pressure and turbid; on examination meningococci were present.

On admission to hospital the patient's pulse rate was 86, his temperature was 38·2° C. (100·8° F.). He was screaming in pain and was quite delirious, head retraction was marked, Kernig's and Brudzinski's signs were present. Herpes was present on lips and nose. Lumbar puncture under general anæsthetic yielded fluid under increased pressure and turbid; fifty cubic centimetres were removed and were replaced by thirty cubic centimetres of antimeningococcal serum. This was done every night until May 14, 1930.

On May 11, 1930, the cerebro-spinal fluid was clear and under slightly increased pressure. No meningococci were present.

On May 12, 1930, an urticarial rash appeared on the body and face, the hands were swollen. All symptoms were now gone except for slight stiffness in the neck muscles. The patient had a big appetite. Incontinence ceased on May 8, 1930. The bowels were constipated, but were kept open with sulphate of magnesia.

On May 14, 1930, lumbar puncture was suspended; the cerebro-spinal fluid was clear and not under increased pressure, no meningococci had been present in the last four specimens. On May 17, 1930, he complained of headache and pain in the back of the neck which was relieved by an aspirin, phenacetin and caffein powder; the headache was worse at night, but the patient passed a fair night with the aid of the powder.

On May 18, 1930, he was much worse, was semidelirious, had no control of urine or fæces and regurgitated fluids through the nose. Head retraction was pronounced. Kernig's sign was present. Lumbar puncture yielded fluid under increased pressure and turbid; symptoms were relieved. On May 19, 1930, lumbar puncture was done, forty to fifty cubic centimetres being removed and replaced by thirty cubic centimetres of antimeningococcal serum. This was repeated every day until June 5, 1930, and then every second day until June 15, 1930. On May 23, 1930, thirty cubic centimetres of antimeningococcal serum were given

intramuscularly and repeated daily until June 24, 1930. On May 30, 1930, a nasal douche of biniodide of mercury was ordered three times a day and continued until June 15, 1930.

From May 18, 1930, the symptoms gradually subsided. Regurgitation of fluids lasted for twenty-four hours. Return of urine and fæces control occurred in three days. Head retraction was gone in ten days; the headache and pain in the back of the neck persisted for three weeks, but were not severe. The cerebro-spinal fluid became clearer and the pressure less, but cocci were present until May 30, 1930.

The temperature was steady on and after June 2, 1930. At no time was it above 38·2° C. (100·8° F.) and the pulse rate varied from 90 to 122.

On July 16, 1930, the patient was quite well and had just left bed with no apparent paralysis or weakness. About three weeks after admission a slight squint was noticed of the right eye; this had not been noticed prior to illness and is still present.

The patient lost a great deal of weight, despite his ravenous appetite, but has now regained most of it. A slurring of speech was noticed during the first four weeks. In regard to lumbar puncture the first four were done under a general anæsthetic, the next six under local anæsthesia, but local trauma was very pronounced and the remainder were done without an anæsthetic.

Features of the Case.

The following are the important features of the case:

1. The mildness of the general signs and symptoms in the early stages.
2. Rapid apparent recovery under serum therapy.
3. Relapse following cessation of the serum therapy.
4. Prolonged period during which cocci were present in the cerebro-spinal fluid.
5. Paralyses: (*a*) Right arm, (*b*) palatal, (*c*) sphincter.

Questions Raised.

The following questions arise:

1. Mode of action of the serum—(*a*) Was it bactericidal? (*b*) Was it antitoxic? (*c*) Was it both?
2. Was there a relapse or reinfection; if the latter, by what path did it occur?
3. Was the supplementary method of the giving of serum intramuscularly of any value?

THE TREATMENT OF DEAFNESS BY THE ZUND-BURGUET METHOD.

By R. H. BETTINGTON, B.A. (Oxon), B.M., B.Ch. (Oxon),
Sydney.

AFTER a small experience, amounting in all to twelve months, I am prepared to make a few remarks on the treatment of deafness with the Zund-Burguet electrophonoid apparatus. Dr. Herbert Marks had my instrument before I took it over and he was pleased with his results, though I heard few particulars from him. I am indebted to Dr. Blashki for original instruction in the use of the instrument.

The very small number of patients in all whom I have treated or am treating, and the short time that has elapsed prevent very definite conclusions being formed, but the results so far are most encouraging. The majority of my patients are suffering from deafness of the middle ear type and I have combined Eustachian catheterization with the Zund-Burguet treatment, until the tubes become freely patent, that is, provided they were obstructed in the first place; it may be argued that the catheter is responsible for the improved hearing, and I think that is partly, but not entirely true. So far, in every case treated for any length of time the heavy sensation described by the patients as "feeling stuffed with cotton wool," when present, has disappeared; this in itself is a great relief and is sufficient reason for treating these patients.

Another feature is the lessening of tinnitus; Case I was the only instance in which it was still present at the end of treatment, and it had improved considerably then.

Case VII I regard as the most satisfactory; the patient was an oto-sclerotic of long standing who feared to go out and meet people; she is now able to converse comparatively easily and has lost entirely the heavy feeling that has been present for so long.

Case VIII is almost too good to be true; that two treatments should have such an effect appears incredible and yet the patient was a very normal type. This case would accentuate the fact that early treatment is essential, if a complete cure is to be obtained. I have other patients under treatment, one with a very marked double nerve deafness, but in no other has enough time elapsed to give any results.

I apologize to any who may read this, for the lack of system in the hearing tests; in future I hope to improve on it.

I am unaware if anyone else besides Dr. Blashki has this instrument; if so, I should be very glad to hear their results.

CASE I.—A female, aged forty-six years, complained of tinnitus in the right ear and heavy feeling in the head. She had a history of frequent colds and had had no serious illness. Hearing was normal on both sides; the right Eustachian tube was very sticky. The family history was nil. Between August 1, 1929, and May 31, 1930, ninety treatments with the Eustachian catheter and the Zund-Burguet apparatus were given to the right ear only; for the last twenty visits the Zund-Burguet apparatus was used without catheterization. Progress was good at first, tinnitus lessened and the heavy feeling in the head was absent; the Eustachian tube became quite patent. With the last twenty-five to thirty treatments there was no progress, tinnitus was still present and very worrying. The patient was sent off for three months' rest.

CASE II.—A female patient, aged twenty-six years, was treated by Dr. Herbert Marks fourteen times. I saw her only twice. She said she had been very deaf and had tinnitus. She had had Zund-Burguet treatment combined with catheterization. The tinnitus was gone and the tubes were patent, the right ear was normal, the left ear could hear a watch at fifteen centimetres (six inches) and the range of tuning forks from 4,028 to 64 double vibrations. The result of the Rinné test was just "negative." She has not been seen since.

CASE III.—A male, aged forty-seven years, with tinnitus in the right ear and a heavy feeling in the head, said that he was deaf in the right ear at times, but on examination his hearing was found to be normal. Twenty-seven treatments with the Eustachian catheter and Zund-Burguet apparatus were given to the right ear; at the close the tinnitus had entirely disappeared and the heavy feeling had departed from the head. The patient was using a menthol and adrenalin spray which probably influenced the result.

CASE IV.—A boy, six years old, born deaf and dumb, had his tonsils and adenoids removed by Dr. Marks eighteen months ago. When seen, he could hear vibratory noises and could enunciate certain words, none of them well. At the present stage, after sixty-three treatments, he can say more words with slightly better enunciation, but there is very little indication of improvement in his hearing; his mother thinks there is a slight improvement, but nothing definite.

CASE V.—A male, aged forty years, gave a history of increasing deafness for six months; there was no family history. When he was first seen on January 6, 1930, with the left ear no tuning fork could be heard by air conduction; by bone conduction forks from 4,096 to 512 vibrations could be heard. From there with forks down to 64 vibrations nothing could be heard; a very loud speaking voice was required. The Eustachian tubes were blown through, but were found to be patent; this treatment was thus not continued. On February 28, 1930, after twenty-one treatments three times a week, both ears could hear by air conduction all ranges of forks down to 64, the right ear

could hear them slightly longer than the left. Bone conduction was the same as air conduction.

On April 16, 1930, after ten more treatments, the left ear improved slightly, the right remained unaltered. He has a very slight disability now; he can hear the ordinary voice at a distance of 300 centimetres (ten feet). On July 7, 1930, he came back after three months' rest; there was no alteration and he has resumed treatment. He says that his hearing has improved vastly and that his friends comment on it.

CASE VI.—A male, aged forty-six years, had complained for twelve months of deafness in both ears. There was a variation in each side at times, but it tended to get worse. He used to get frequent colds and even now has a lot of discharge from the nose. The ears discharged on and off for years when he was a boy. The right tympanic membrane had a small dry anterior perforation and forks from 4,096 to 128 double vibrations could be heard easily; fork 64 could not be heard. The left tympanic membrane was absent. The ear was dry and forks 4,096 to 512 could be heard. The result of Rinné's test was "negative" on both sides. The nasal mucous membrane was congested and both Eustachian tubes blocked, especially the left. After sixteen treatments by catheterization and the Zund-Burguet apparatus to both ears, also menthol and adrenalin drops, the right tube is clear, the left is still sticky, the nasal mucous membrane is much healthier, the right ear can now hear fork 32 d.v. Bone conduction about equals air conduction. The left ear hears fork 128 d.v. well. Catheterization was discontinued on May 3, 1930.

On July 4, 1930, the right ear could hear fork 32 d.v., the left ear fork 64 d.v. The tubes were patent. Bone conduction now equals air conduction. The patient has been sent away for two months' rest, his head feels much less stuffed up and he says that he can now hear all ordinary conversation.

CASE VII.—A female, aged fifty-eight years, was first seen on April 1, 1930. She complained of deafness for twenty-five years; the onset was sudden after the birth of her second child. Her condition has been stationary until about four months ago, since when it has become more pronounced. The left ear is worse than the right, there is no tinnitus, there is no family history. The head feels very heavy and stuffed up. The meati are wide, no wax is present, the tympanic membranes are both opaque and slightly retracted. The Eustachian tubes are patent. The right ear hears from fork 4,096 d.v. down to fork 512 d.v. by air conduction. The left ear hears from fork 4,096 d.v. to fork 1,024 d.v. by air conduction. The result of Rinné's test is strongly "negative" on both sides. After eighteen treatments on May 20, 1930, the right ear hears fork 256 d.v. by air conduction and the left fork 435 d.v. by air conduction. The head heaviness has now disappeared. The patient feels much less deaf and can face strangers without her previous trepidation; she has had to ask her family not to speak so loud.

CASE VIII.—A female, aged forty-five years, consulted me on June 4, 1930. She complained of two months' deafness in the left ear. It came on suddenly and varies in intensity; there is slight tinnitus of a ringing nature, the left ear hears forks by air conduction from 4,096 d.v. to 256 d.v., below that it cannot hear by air or bone conduction. It hears a watch at six millimetres (a quarter of an inch). The left Eustachian tube was slightly obstructed, it was blown through, but there was no immediate improvement in hearing. After two treatments only she can now hear forks down to 32 vibrations well and can hear the same watch at 22·5 centimetres (nine inches) from the left ear and only 17·5 centimetres (seven inches) from the right.

CASE IX.—A female, aged forty-three years, was deaf in the right ear for two years. It came on suddenly when she had a cold; there was no family history, tinnitus was marked. The left ear was normal. The right ear heard forks from 4,096 d.v. to 256 d.v., bone conduction was lessened, the right Eustachian tube was patent. After nineteen treatments tinnitus has gone, but there is no change in the results of hearing tests. After twenty-eight treatments fork 128 d.v. is just heard. She says she feels no change in hearing, but is much relieved by the cessation of tinnitus.

CASE X.—A male, aged fifty-five years, had a history of twenty years' deafness; this had not increased in the last ten years. There was a gradual onset. No family history was obtained. The patient used to have tinnitus, but has none now. There was a history of nasal obstruction following injury. The patient had a submucous resection of the septum done fifteen years ago, which caused temporary improvement. The nasal passages are clear, the Eustachian tubes are patent. A little wax is present in the meati, the drums are retracted, the promontories are visible. The left ear hears forks from 4,096 d.v. to 256 d.v. and right ear from 4,096 d.v. to 128 d.v. Bone conduction is slightly greater than air conduction on both sides. After seven treatments the left ear is the same and the right hears down to fork 64 d.v. Unfortunately the patient has had to go away, but says that he feels less deaf and easier in the head than before.

Reviews.

THE TONSILS AND TONSILLAR SURGERY.

"TONSIL SURGERY," by Robert H. Fowler, M.D., is a book of 288 pages, printed in clear type and profusely illustrated.[1] The author maintains that he presents the subject in a different way, in that he makes special reference to the anatomy of the region, describing as a new muscle a part of the *palatopharyngeus* which is attached to the tonsil capsule, and giving a special name to it—*tonsilopharyngeus*. It is doubtful if this attachment of muscle fibres of the *palatopharyngeus* to the tonsil capsule deserves a separate name. It has generally been recognized by surgeons that some muscle fibres of the *palatopharyngeus* are attached to the tonsil at the junction of the upper and lower lobes. He also states that the tonsils fade out on the posterior pharyngeal wall and that there is no well defined posterior pillar. This is not generally the case, for as a rule one can find definite evidence of a posterior pillar of the fauces to be present. He also denies that the tonsil has any capsule, regarding this so-called structure as just a thickened layer of fibrous tissue reinforcing the submucosa of the second pouch. However, throughout the book he refers to the capsule of the tonsil.

The *plica triangularis* is described in certain cases as being composed of two parts, *pre-* and *infra-triangulares*. The muscle relations of the tonsils are very well described and the illustrations of different dissections are very clearly shown.

Stress is made on the proximity of the glosso-pharyngeal nerve to the lower pole.

Altogether this chapter on the anatomy is very good and gives one a very thorough understanding of the structure in this region.

The chapter on the structure of the tonsil goes to explaining the formation of the tonsil as a pharyngeal pocket lined by exuberant lymphoid tissue which obliterates the central part of the cavity.

Nothing new is suggested as to the physiology of the organ. We agree with the author when he states that he considers the statement that "sick people of all ages show 60% infected tonsils" as being a low figure, and also when he states that "when a tonsil becomes infected its defensive properties are gone."

The case of focal infection is also very clearly described, the indications and contraindications for tonsillectomy being clearly put. He regards as contraindications high blood pressure, bleeders and pregnancy. He considers that when there is a doubt as to whether an operation will be worth while it is better to be conservative. Under the heading of classical operations he gives five different methods of dissection, including one specially described as the author's operation. This is a very clear description;

[1] "Tonsil Surgery, based on a Study of the Anatomy," by Robert H. Fowler, M.D.; 1930. Philadelphia: F. A. Davis Company. Demy 4to, pp. 288, with 103 illustrations, including ten full-page colour plates. Price: $10.00 net.

in it he points out the advantage of saving flaps of mucous membrane to help to cover the raw area. The work of George E. Waugh on the dissection method is entirely omitted.

In a chapter on automatic instruments Sluder's modification of the guillotine method is described, but the earlier work by Willis and Pybus receives no mention at all. The reader feels that the author is not so conversant with this method of tonsillectomy as he is with the dissection method. He quotes equally emphatic views of equally able surgeons advocating removal of the tonsils by means of the guillotine in one case and by dissection in the other. The author states his own view thus: "The training of the throat surgeons should include experience with both methods to make clear the good and bad points, their value and their dangers."

In a chapter on complications a very interesting list is given. Out of thirty-three fatalities occurring within twenty-four hours of the operation, eight were caused by the local anæsthetic, three by the general anæsthetic, fourteen were caused by hæmorrhage, but it is not stated the proportion associated with general and with local anæsthesia.

He considers aspiration to be the chief cause of pulmonary abscess following tonsillectomy, but no definite statistics regarding the frequency of this complication are given. In the discussion of the less serious complications stress is laid on the dangers of leaving pieces of tonsillar tissue. The worst cases of cardiac disease, rheumatism and nephritis are found where only a partial operation has been done. More serious results follow when portion of the upper pole is left. This is a recognized observation which cannot be too strongly stressed.

Regarding the effect of tonsillectomy on singers we quite agree with the author that a clean tonsillectomy, leaving the *plica triangularis* and lymphoid nodules below this, does not harm the voice, but usually improves it. That the operation for the removal of the adenoids need not be a complete one, does not appear to be in accordance with the general opinion. One frequently sees chronic Eustachian catarrh and nasal sinusitis associated with infected remnants of adenoid tissue left by previous operations.

In a chapter on hæmorrhage the difference between the true hæmophiliacs and bleeders is pointed out. The coagulation test is of no value in determining the clotting of the blood. In severe secondary hæmorrhage he does not hesitate to tie the external carotid and its branches. Both external carotid arteries may be tied. He has tied the common carotid five times and two of the patients died.

The technique of general anæsthesia is very inadequately described. That for local anæsthesia is well and clearly given.

Under "Follow-up Reports" the author points out that 93% of the parents of 1,200 children under fourteen years said they were glad of the operation. In one hundred adults equally good results were found in cases of sore throat, rheumatism and frequent colds.

A chapter on evolution goes very deeply into the subject. An extensive bibliography is given at the end of the book. Altogether the author has produced a very interesting volume. Surgeons practising this specialty will find it most useful. The fact that the anatomy of the region is so excellently shown adds very materially to its value.

OPHTHALMOLOGY.

"A MANUAL OF DISEASES OF THE EYE," by Dr. Charles May and Dr. Claud Worth, is too well known to require any introduction.[1] Its value to the student and to the practitioner is further demonstrated by the appearance of its sixth edition. This is a fuller proof of its utility, when we recall that it is the successor of a manual prepared by Dr. May which had reached four editions prior to the appearance of the joint production.

The various methods of examination are clearly described. The ease with which the novice can learn the execution of these methods is increased by the series of photographs depicting the various stages. The coloured plates representing external diseases and fundus lesions are of great value to the student of ophthalmology. The latter, though they lack the artistry characteristic of the illustrations in certain other text books, do not lose thereby in utility. By being partly diagrammatic the typical features of the various fundus conditions are emphasized and thereby impressed more clearly on the mind of the student.

The section on comitant squint is dealt with clearly and with an authority that is justifiable, because of Dr. Worth's unique experience in the non-operative treatment of squint by fusion training. Moreover, his is largely the credit of establishing in English speaking countries the superiority of the operation of advancement.

Sections dealing with colour vision and ocular microscopy are of value to the student who is about to specialize. They are written by Mr. T. Harrison Butler and the late Dr. Devereux Marshall.

This is a book that we can thoroughly recommend to the medical student and practitioner. Both will be aided in the recognition of the common conditions by the clear and concise manner in which the author emphasizes the essentials for diagnosis.

A RÉSUMÉ OF OBSTETRICS AND GYNÆCOLOGY.

BOOKS of the style of "Synopsis of Midwifery and Gynæcology," by Aleck W. Bourne,[1] have somewhat limited uses; they are essentially "cram" books. As the author endeavours to cover the whole field it is not necessary to describe in detail the various divisions of his book. Reference will be made to only two or three points.

The statement that pregnancy tends to be lengthened when the woman is able to take much rest during the later months can be proved in any indoor antenatal department where many women go over their estimated date. These women do very little work and their days are spent in sitting and lying about.

The urine should be examined every week and not once a fortnight during the last month. The blood pressure should also be taken.

The author states that chloroform should not be used in eclampsia, but also mentions that it is used by Stroganoff in his treatment; many people use chloroform in eclampsia with success. In discussing diabetes the author does not mention "Insulin," which now considerably alters the aspect. In the treatment of contraction ring no mention is made of an injection of adrenalin. We are pleased to see the author state that it is the exhausted condition of the patient that makes *post partum* hæmorrhage a great danger in the third stage in *placenta prævia*. In the treatment of inversion of the uterus no mention is made about the removal of the placenta, whether to peel it off before replacing the uterus or to replace the uterus with the placenta still attached and remove it afterwards.

The author has advocated the earliest possible surgical interference in acute salpingitis. There are some very eminent men who are opposed to this treatment.

No operative details are given. The teaching on the whole is very sound and the author himself states many of the best known text books have been consulted. For the student working for an examination or anyone requiring a summary of midwifery and gynæcology, the book will be found of use.

[1] "A Manual of Diseases of the Eye," by Charles H. May, M.D., and Claud Worth, F.R.C.S.; Sixth Edition; 1930. London: Baillière, Tindall and Cox. Demy 8vo., pp. 483, with 22 coloured plates. Price: 15s. net.

[1] "Synopsis of Midwifery and Gynæcology," by Aleck W. Bourne, B.A., M.B., B.Ch. (Cambridge), F.R.C.S. (England); Fourth Edition; 1929. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 441, with illustrations. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 13, 1930.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

RESEARCH AND THE COMMUNITY.

THE average member of the community—"the man in the street"—looks upon people engaged in scientific research and even on those who earn their bread and butter by teaching the basal sciences, as somewhat peculiar beings dealing with abstract matters that are not of much use to anybody. Others attribute to the scientific worker almost supernatural powers in the discovery of the secrets of Nature. Individuals of the first type give little thought to the results of research, even when these are forced upon them, and those of the second type are prone to regard the scientist as somewhat of a failure unless he justifies his existence by producing results. Relatively few people view scientific research in a proper perspective. Unfortunately those who do not understand are the most blatant in the public press and the most noisy on the public rostrum; by their clamorous persistence they carry with them others who will not take the trouble to think for themselves.

At the present time the Government of Australia does not devote a great deal of public money to medical research; it has no large annual appropriation of funds analogous to that provided every year for the Medical Research Council of the Privy Council of Great Britain. It does, however, make provision for the needs of the Council for Scientific and Industrial Research. The activities of this council are directed along six channels. The six "divisions" are those of animal nutrition, animal health, entomology, plant industry, soils and forest products. It would be difficult to overstate the importance of the study of these subjects to Australia and its primary industries; for the readers of a scientific journal it is not necessary to make the attempt. There would be no necessity to refer to the subject were it not that one section of the daily press has brought forward the suggestion that, owing to the present state of financial depression, the activities of the Council for Scientific and Industrial Research should be suspended. No thinking person will believe that those responsible for this suggestion do not realize the work that the Council has done, and do not know what it is doing. We are given to understand, for example, that the prickly pear problem is nearing solution and no great imagination is needed to picture the enormous economic gain that would result from such an achievement. The only possible conclusion is that the newspaper suggestion is made as a sop to the blatant individual already mentioned. The present Government of the Commonwealth is fully alive to the value of the work of the Council for Scientific and Industrial Research and has no intention of interfering with it. Governments, however, sometimes feel themselves compelled to heed a popular outcry and herein lies the danger at the present time.

The situation may be met only by creating a body of public opinion, educated to a proper appreciation of the difficulties and the disappointments of research and cognisant of the need for dispassionate evaluation of its results. Professor O. U. Vonwiller, of the University of Sydney, recently gave expression to views that should be broadcast among scientists. Speaking on behalf of the Royal Society of New South Wales at the annual dinner of the Chemical Society of the Technical College (Sydney), he said that scientists themselves were possibly to blame for some of the misconceptions prevalent in the minds of the public. He thought that scientists

laid too much stress on their successes and he deprecated the fact that they sometimes promised that certain results would follow at once if support were forthcoming. With his plaint we are in entire agreement. As far as medical investigators are concerned, it is too often noted that a procedure successful in one direction is applied without justification or with but slender justification in others and that quite unwarranted claims are made for it. A scientist's success must be tempered with humility. The public should be made to understand that for one set of observations of practical utility many channels have been explored which apparently lead nowhere, that there have been scores, possibly hundreds, of abortive experiments, that a gain is often made by piecing together scraps of information gleaned from apparently unconnected sources, that research directed towards preconceived explanations and conclusions is worthless, that research may be the occasion of much mental stress and anguish, and that failure is more usual than success. When this point of view has been presented, by all means let emphasis be laid on past achievement, let attention be drawn to economic gain, and let the lives of thousands saved to the community be counted. A proper perspective will have been attained. In no other way is it possible to prevent the setting in of the rot that threatens to destroy the fabric of a valuable structure; if the rot is allowed to start, there is no knowing where it will stop.

Current Comment.

HYPERPARATHYREOIDISM.

THE epithelial bodies derived from the third and fourth pharyngeal pouches in the embryo form the structures known in the adult as the parathyreoid bodies or glands. The activity of these glands, associated with the presence of vitamin *D*, is essential to calcium metabolism in the animal economy. In the absence or marked deficiency of parathyreoid activity, tetany develops. Tetany is intimately concerned with the presence of guanidine and its derivatives, particularly methyl-guanidine. Possibly parathyreoid secretion detoxicates guanidine and its compounds. At the present time the public, owing to extensive advertising on the part of enterprising chemical firms and easy access to the preparations, dose themselves in wholesale fashion with calcium compounds together with parathyreoid extracts for such conditions as urticaria and, in the winter, pernio or chilblains. Accordingly, the sinister possibilities of parathyreoid overdosage cannot be too strongly emphasized.

Collip discovered an active parathyreoid hormone in 1925; this he called parathormone. It is now possible to study hyperparathyreoidism experimentally. The characteristic feature of excessive parathormone injection is hypercalcæmia, sometimes exceeding twenty milligrammes per hundred cubic centimetres of serum. Hypercalcæmia is of rapid development, attaining a maximum in from twelve to twenty-four hours, but rapidly falling. Concomitant symptoms are restlessness, respiratory distress, vomiting, diarrhœa, melæna, hæmatemesis, collapse and death. Hæmaturia is common and anuria may supervene when the blood calcium is at its maximum, some hours before death. Towards the end inorganic phosphorus, urea and non-protein nitrogen of the blood rise rapidly. Chlorides are frequently diminished. The blood plasma is decreased in volume, with increased viscosity. Coagulation time is lessened. There may be marked acidosis. Necropsy reveals hæmorrhages into the kidneys and the walls of the stomach and intestines. Large amounts of calcium are found in the kidneys, heart and liver, as well as in the blood. Increased excretion of calcium and phosphorus creates a negative balance of these elements. Greenwald and Gross (1925) gave to dogs sufficient parathormone to cause death in six days. They noted not only hypercalcæmia, but also a great increase in the excretion of calcium and phosphorus, which increased independently of the greater protein metabolism. Further experiments were conducted with smaller doses over a greater period. It was concluded that an abnormally high excretion of calcium and phosphorus could be maintained for a long time and that the calcium must be derived from the bones. Hueper (1927) observed, from parathyreoid overdosage, metastatic calcification in the lungs, kidneys, stomach, duodenum, heart muscle and thyreoid gland, with calcium casts in the tubules of the kidneys, small hæmorrhages in the brain and thrombi in the small vessels of the myocardium with areas of localized necrosis. The hormone alters calcium distribution in the body, abstracting it from some tissues, such as bone and muscle, and heaping it up in others, as the kidneys. These findings very closely resemble those produced by vitamin *D* poisoning when excess of irradiated ergosterol is given. Gordon and Cantarow (1927) found that parathormone administered preoperatively in jaundice reduced the coagulation time of the blood to normal, but, with large doses over a prolonged period, bleeding seemed to increase. In the hypoparathyreoidism of tetany there is increased muscular irritability and tonus. With parathormone excess hypotonicity and diminished irritability would be expected.

D. P. Barr and H. A. Bulger, after an account of experimental hyperparathyreoidism, give details of

all the parathyreoid tumours reported to date and add some cases of their own.[1] Parathyreoid tumours have been recognized since Kocher's report in 1899. Up to date, seventy-four examples of pathologically enlarged parathyreoid glands have been recorded, being examples of simple hyperplasia and benign and malignant tumours. Malignant growths are rare and have not been associated with evidence of functional derangement of the parathyreoids or with important bone conditions. Some of the conditions associated with parathyreoid enlargement are merely incidental; others, such as nephritis, are so frequent that they must be considered as the result or the cause of the parathyreoid changes. Most conspicuous of all in this connexion are diseases of bones, such as a form of osteomalacia, giant-cell tumour of bone, and especially generalized *osteitis fibrosa cystica* (von Recklinghausen's disease). This disease is usually seen in adult life, but may start in childhood. One case has been recorded in an old man. Most of the cases occur in females. The cysts and tumours principally affect the long bones, but the bones of the skull, the jaws and the pelvis may be implicated. The condition may escape recognition until a fracture (a very frequent occurrence) directs attention to it. The femur and humerus are the bones most often fractured, with accompanying pain. The cysts may be the site of extensive hæmorrhages and fibrosis. Owing to the great number of giant cells in the fibrous tissue, a giant-celled sarcoma-like epulis may be simulated. Occasionally the bone cyst is solitary in the humerus or tibia. There may be general or localized decalcification—a form of osteomalacia. Schmorl and others consider that the disease has no relationship to puerperal osteomalacia or to rickets. Instances of *osteitis fibrosa cystica* have been recorded with no obvious parathyreoid lesion, but tissue anatomically normal may have increased or altered functional capacity. Barr and Bulger quote a case of multiple myeloma with hypercalcæmia but no other manifestation of parathyreoid disease. Possibly administration of large doses of calcium lactate may influence the level of blood calcium, but this has not been satisfactorily established. Serum calcium may be increased by irradiated ergosterol, but not after removal of the parathyreoid glands. High values of serum calcium have been reported in gout, *arthritis deformans*, other arthritic conditions and in *polycythæmia vera*, but the evidence is conflicting. The belief that the hypercalcæmia of von Recklinghausen's disease is due to parathyreoid over-activity rests on the fact that some of the concomitant symptoms (increased urinary excretion of calcium, abnormal deposit of calcium in the tissues and muscular weakness) are seen in experimental parathormone overdosage, upon finding parathyreoid hyperplasia or tumour and upon the fall in serum calcium after removal of such a tumour. But solitary cysts and single giant-cell tumours similar to those of von Recklinghausen's disease may exist without evidence of parathyreoid disease. Multiple fibrocystic disease may exist without hypercalcæmia. Even if parathyreoid symptoms are conspicuous, hyperparathyreoidism may not be the primary factor. Possibly the bone changes occur first and evoke increased parathyreoid activity which then aggravates the bone condition, establishing a vicious circle. In multiple myeloma probably the bone changes are primary and the parathyreoid hyperplasia secondary. It is possible that many generalized diseases of bone accompanied by marked absorption or destruction of bone substance lead to parathyreoid hyperplasia which may assume the predominant rôle in the clinical picture. Removal of the parathyreoid tumour in generalized *osteitis fibrosa cystica* has ameliorated the symptoms; later, with rapidly falling blood calcium, tetany has supervened. Hypercalcæmia should always arouse suspicion of hyperparathyreoidism. Perhaps this hyperactivity may exist without hypercalcæmia. For the maintenance of the serum calcium level vitamin *D* is necessary (or the ultra-violet rays of sunlight). The absence of these essentials is responsible for the low calcium values in rickets. In the rickets of children and animals there has been noted hyperplasia of the parathyreoids which may be compensatory. The serum calcium value in rickets is the resultant of two factors, parathyreoid over-activity tending to raise it and the absence of vitamin *D* tending to lower it.

[1] *The American Journal of the Medical Sciences*, April, 1930.

CARCINOMA OF THE DUODENUM.

ALTHOUGH the stomach is a common site for the growth of cancer, the duodenum is seldom attacked. Many observers believe that ulcer of the stomach is not often followed by malignant disease; others hold an opposite view. Duodenal ulcer is generally believed to carry with it no risk of malignant disease. It is of interest, therefore, to read a recent report by E. Ewart Schofield.[1] His patient was a married woman, aged thirty-five years, who gave a history of epigastric pain, not severe, but extending over a period of several months. The pain was not related to the taking of food. Vomiting had occurred on and off for three months. Jaundice was present for about two months and the depth of jaundice varied from day to day. A lesion in the soft palate suggested syphilis, but the Wasserman test yielded no reaction. A diagnosis of carcinoma of the pancreas was suggested. At operation a pear-shaped sessile ulcer, about three centimetres long, was found on the posterior wall of the duodenum; the proximal end encroached on the ampulla and caused partial obstruction to the flow of bile. A piece of the ulcer removed for examination was found to be adenocarcinomatous. In his conclusions Schofield states that duodenal ulcer has not been proved to be a predisposing cause to duodenal cancer.

[1] *The British Journal of Surgery*, July, 1930.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Treatment of Menopausal Symptoms.

J. SAKHEIM AND H. LEHFELDT (*Monatsschrift für Geburtshülfe und Gynäkologie*, March, 1930) discuss the blood picture at the menopause. There is often a relative degree of lymphocytosis which the authors believe to be due to varying degrees of thyreoid toxicosis. In the treatment of the various symptoms at this period of life they recommend venesection at regular intervals and the use of saline purgatives, especially sodium sulphate, with occasional doses of calomel. Bromides are given usually in a form suitable for intravenous administration.

The Curette.

H. E. STEIN (*The Medical Journal and Record*, April 16, 1930) criticizes the use of the sharp curette and advocates the abolition thereof from the armamentarium of the gynæcologist as well as the general practitioner. For abortion he considers that a blunt instrument is all that is necessary; or even ring forceps can be used to do all that is required. The author considers the curette ineffective in hydatidiform mole and advocates primary hysterectomy on account of the malignancy of the condition. He deprecates the curetting of a uterus merely because of menorrhagia or metrorrhagia or leucorrhœa. When used for diagnostic purposes to remove scrapings of a suspected malignant growth or polypus there may be justification, but it is quite possible to miss the malignant area and so fail to obtain the desired evidence.

The Function of the Cervix During Coitus.

A. WALTON (*The Journal of Obstetrics and Gynecology of the British Empire*, Spring Number, 1930) describes a series of experiments upon rabbits to determine the effect of the cervix upon fluids in the vagina. It has been claimed that coordinated movements of the cervix and body of the uterus have a direct action in drawing into the uterus the seminal fluid. Female rabbits in the follicular stage of the œstrous cycle were injected *per vaginam* with various coloured solutions such as gelatine-Ringer coloured with methylene blue and indian ink and a graphite suspension and immediately afterwards were mated with vigorous males and at definite intervals the animals were killed and the genital tract examined. The results go to show that there is no tendency for the fluids to be drawn into the uterus as a result of coitus. It is laid down that the spermatozoa gain entrance entirely because of their own motility and that the cervix acts rather as a valve to prevent the forcible entry of any fluid into the uterus, it being possible to rupture the vagina with fluid under pressure before any will gain entrance to the cervical canal.

Endocervicitis.

IN dealing with the subject of treatment of endocervicitis with the actual cautery and electrocoagulation, Walter J. Harriman (*American Journal of Obstetrics and Gynecology*, August, 1929) states that he has treated two hundred patients suffering from endocervicitis by cauterization and prefers electrocoagulation to the actual cautery. The treatment varies with the degree of cervical involvement. In the milder forms of the condition the canal is "striped" or coagulated by linear coagulations in four or five sections. In the more involved cervices the electrode remains in contact with any certain area of the cervical canal for a longer period of time and the electrode is drawn more slowly from the internal to the external *os* just fast enough to prevent arcing or sparking of the current. The erosion on the vaginal surface is treated in the same way until the entire area has become white —a proof of its coagulation. The diathermy machine is previously adjusted by plunging the active electrode into a square inch piece of veal or liver resting on an indifferent electrode. The lowest milliampèrage is read and is the standard employed for the cauterization of cervices. Coagulation of the tissue at the point of contact occurs in one second.

Urethral Caruncle in the Female.

CHARLES T. OLCOTT (*Surgery, Gynecology and Obstetrics*, July, 1930) reports a series of investigations into the condition of urethral caruncle in the female. He points out that the epithelium of the urethra shows considerable individual variations, being either squamous or transitional in type. Various authors have found these distributed in different ways along the lumen. He considers that the epithelium of the inner end of the urethra resembles that of the bladder, while at the meatus he has found both squamous and transitional epithelium sometimes in the same transverse section. Urethral caruncle is of the nature of a granuloma and consists partly of hypervascular, papillary or telangiectatic mucoid polypi. Ewing has pointed out that there are normally invaginations of the epithelial lining of the caruncle which on section may appear as displaced islands of atypical epithelium, strongly resembling epithelioma. A review of the literature of those caruncles which have been reported, does not throw any light on the ætiology. Childbirth, apparently, chiefly through its relation to infection, and gonorrhœa both seem to predispose. They seem to be more common about the menopause. The author has studied twenty-three specimens which include nineteen from the New York hospital. The ages of the patients vary from twenty-three to fifty-five, the average being fifty-one years. No history of venereal infection occurred in the series. Pathological examination of the series showed that in ten of the twenty-three cases there was an equal amount of stratified or transitional epithelium. In six cases there was apparent epithelial infolding, arousing a suspicion of malignant disease. In seventeen of the twenty-three cases there was definite glandular tissue in the submucosa. The author believes them to represent Skene's glands, as they have a compound acinar structure similar to those glands. They suggest that these glandular structures may be an important factor in the formation of caruncles.

The Treatment of Prolapsus Uteri.

THE treatment of *prolapsus uteri* according to the methods of the Manchester school is described by William Fletcher Shaw (*Proceedings of the Royal Society of Medicine*, June, 1930). Shaw is the advocate of the method of the Manchester school as initiated by Donald in 1888 and followed up by Fothergill. His paper is a brief review of the whole question of *prolapsus uteri*. After dealing briefly with causation, symptoms and general principles of treatment, he details the colporrhaphy as performed in his own clinic. The paper is illustrated with twenty diagrams. The results of his work in 1,828 patients operated on gives an operative mortality of 0·43% and an absence of recurrence in 90%. The complications that have been met with in his series are retention of urine, occasional hæmorrhage about a week after the operation, rarely occlusion of the cervix, occasional vaginal adhesions; while he regards ulcers of the cervix as a definite contraindication to operation and strongly urges that the patient should be kept in bed with alkaline and astringent douches for at least three weeks before operation, to allow any ulcers of the prolapsed mass to heal.

OBSTETRICS.

The "Physiological" Anæmia of Pregnancy.

P. BROOK BLAND, LEOPOLD GOLDSTEIN AND ARTHUR FIRST carried out a study on a thousand cases in regard to the so-called "physiological" anæmia of pregnancy (*Surgery, Gynecology and Obstetrics*, June, 1930). They found that 47·4% gave evidence of an anæmia with an erythrocyte count of 3,500,000 per cubic millimetre or less. In 58·6% there was a distinct lowering of the hæmoglobin value below 70%. They found, however, that 56% of the patients in the last three months of pregnancy had a moderate to a severe type of anæmia, while only 24·7% of the patients in the first six months of pregnancy showed the same degree of anæmia. They are of opinion that the anæmia is as a rule more pronounced with the advance of pregnancy. The anæmia, however, commences to

improve rapidly after child-birth. Great improvement was found within seven to ten days after confinement in 72% of the patients who were profoundly anæmic during pregnancy. Within two to six months after delivery there was a very great improvement in the blood count, in many instances a little over the normal figures being found.

Still-Birth Due to Intracranial Injury.

JOYCE PARTRIDGE (*The Journal of Obstetrics and Gynæcology of the British Empire*, Spring Number, 1930) presents a series of cases of women examined prenatally and followed through to their delivery. The cases are presented in tabular form under the following heads, as regards the mother: age, parity, measurements, week of examination, and degree of movement possible at the pubic joint; as regards the infant: weight, presentation and position, method of delivery; and as regards labour: the duration of the first and second stages. She states that nearly half the infants who are alive at the commencement of labour and yet are born dead to healthy mothers, die solely because they have sustained cerebral birth injuries. Relaxation of the ligaments of the pelvic joints by such manœuvres as the use of Walcher's position is an aid to parturition. As regards age of the mother, it is asserted that the years of age during which the normal range of movement of the pelvic joints of the female is greatest, correspond to those during which the percentage of still-births due to disproportion is smallest. In the pregnant woman an increase in this normal mobility of the joints can easily be recognized during the twenty weeks before delivery. Of four patients reported to have no movement at the pelvic joints, two had still-births.

The After-Effects of Multiple Pregnancy.

N. A. GIDALEWITSCH (*Monatsschrift für Geburtshülfe und Gynäkologie*, March, 1930) has reviewed in detail the complications arising in the puerperium in cases of multiple pregnancy as compared with those following single births. The series comprised 564 cases in 36,700 deliveries. He states that the percentage of complications in multiple pregnancies is very noticeable. The number of morbid conditions is three times that noted with single births, while the mortality is ten times as great. No great difference exists between premature and full time deliveries in these cases. The percentage of operative deliveries was much higher and the morbidity following such methods was twice that of the natural deliveries. Subinvolution of the uterus was very common.

Eclampsia.

G. W. THEOBALD (*The Lancet*, May 24, 1930) discusses the theories at present accepted as to the causes of eclampsia and reports experimental work carried out at Bangkok. He denies that eclampsia is caused by any toxin of placental origin or by any functional or organic lesion of the kidneys. The defensive forces of the body are compromised by pregnancy in many ways; the liver has to undertake duties both for mother and fœtus; the anxieties associated with pregnancy may set up nervous influences which interfere with proper digestion of food, as also does the bulk of the uterus; the fœtus requires large quantities of sugar, about eight grammes of calcium and corresponding amounts of other substances during the last four weeks of pregnancy. Deficiencies of the mother's diet are exposed by pregnancy. Intestinal stasis aggravated by the bulk of pregnant uterus and by the fixing of the fœtal head in the pelvis causes an increased absorption of toxins. A lean meat diet fed to dogs causes severe changes in the liver and kidneys. Such a diet contains all the vitamins, but little calcium. This diet, if given to bitches early in pregnancy, will cause death of the fœtus, and human abortions for which no other reason can be given, may be caused by dietetic deficiencies. Sunlight, absence of worry and a diet having a low protein content go hand in hand with a low incidence both of the toxæmias of pregnancy and diseases of the intestinal canal. Eclamptic blood is not particularly toxic to pregnant bitches. Sodium and potassium oxalate given to dogs will cause them to die in convulsions, but the animals may be saved by the intravenous injection of calcium. Guanidine carbonate causes a rapid and prolonged rise of blood pressure. It has a very much more toxic effect if the blood calcium be previously lowered. If intestinal stasis is set up by closure of the anus, the liver and kidneys become severely damaged, whether the animal be pregnant or not, but the results occur more quickly in the pregnant animal. Parenchymatous degeneration of the kidneys is not necessarily associated with albuminuria. The damage to the kidneys is slight when compared to the damage to the liver. It is probable that calcium is the chemical most closely concerned with protecting the body from toxins and that it can exercise such functions only in an ionized form. The available maternal supply of calcium may be deficient for several reasons, such as deficient intake, deficient absorption, increased elimination, the amount required for making the colostrum, the large amount required by the fœtus, and because of vitamin *D* deficiency. A damaged liver cannot properly metabolize meat, so that guanidine is increased and this is antagonistic to calcium and lowers blood sugar. A damaged liver is associated with deranged metabolism in many ways; failure to produce antithrombin would allow fibrinogen to become excessive and cause hæmorrhages. The fits may be caused by sudden drop in blood sugar associated with hypocalcæmia, by œdema of or hæmorrhages into the brain, or by toxins acting directly on the central nervous system. The author's thesis is that eclampsia is caused by toxins absorbed from the intestinal canal which, owing to a breakdown in the defences of the body, are not detoxicated. He further considers that a deficiency of ionized calcium in the blood is the cause of the breakdown of the defences of the body.

Liver Extract in Toxæmias of Pregnancy.

J. L. RYECRAFT (*The American Journal of Obstetrics and Gynecology*, June, 1930) reports a series of fifty patients with toxæmia treated by the injection of liver extract in the form of "Heparmone"; in some cases glucose was given intravenously as well. In all, the histories of fifty patients are summarized; they are divided into three classes according to their blood pressure and their urinary condition. In three the condition was mild, in twenty-seven it was moderately severe and in twenty it was severe. One patient who was on the verge of eclamptic seizures and who chanced to come to autopsy, had had a very large amount of "Heparmone" and her liver revealed no degenerative changes. Of ten patients who actually had eclampsia, eight were delivered of living babies. The author thinks that the blood sugar curve is of prognostic value and gives glucose in addition to liver extract to all patients who are thought to be in a preeclamptic state. The liver extract used contained a minimum of nitrogenous matter and is used in addition to the usual measures, such as sedatives and elimination.

Complete Lacerations of the Perineum.

L. E. PHANEUF (*The Medical Journal and Record*, April 16, 1930) discusses the prophylaxis and treatment of perineal lacerations. Prophylaxis is divided into two classes, first by manually dilating the vagina and perineum, and secondly by cutting the tissues in such a place as to cause least damage in the event of the tear spreading. Perineotomy is recommended when the disproportion between the presenting part and the vaginal outlet is not too great. Episiotomy or lateral perineal incision should be resorted to when a great deal of room is required, as in difficult forceps deliveries. If primary healing of the repair done immediately after delivery does not occur, at least three months should be allowed to elapse before secondary repair is undertaken. The author describes the technique of the repair operation, in which he makes use of an H-shaped incision. The after-care recommended by him requires the patient to have the bowels closed for six days. The diet consists of albumin, orange water and water for five days, with beef tea or broth on the sixth. Thirty cubic centimetres (an ounce) of castor oil are given on the seventh and an olive oil enema. Catheterization of the bladder every twelve hours is resorted to if the patient does not pass urine regularly.

Special Articles on Diagnosis.

Contributed by Request.

XI.

PRECORDIAL PAIN.

WHEN investigating the condition of a patient suffering from precordial pain, three facts must be considered:

First, the date of onset. This may be easily elicited, or it may require a considerable amount of cross-questioning of and consideration by the patient.

Secondly, the conditions which make the pain worse. These are: (i) The relation of the pain to effort, or even the time after effort when the pain comes on; (ii) the relation of the pain to food, sleep and certain movements, for example, bending or turning either to the right or to the left, or breathing.

Thirdly, the case must be investigated anatomically, that is, the mind's eye must pass through the body, structure after structure, starting from the skin over the precordium and coming out at the skin at the other side of the body.

If these things are borne in mind, a diagnosis of considerable accuracy can usually be made.

Anatomical Considerations.

We must now consider the patient anatomically.

A skin condition which may cause precordial pain, is herpes. In the acute stages the pain may last for some days before the rash comes out, and the condition is very difficult to diagnose before the rash appears; but the pain has a tendency to go right along the course of a nerve. Post herpetic neuralgia is unusual in this situation, but when it occurs, the pain is constant and aching. It is not its severity that drags the patient down, but its constancy. Careful examination of the skin will show in practically all cases post herpetic scarring, and the patient, on being questioned, will probably remember that he has had a rash over this area.

The next structure we come to is that of the muscles. Two conditions affecting the muscles which give rise to precordial pain, are muscle strain, causing rupture of the muscle fibres, and muscular rheumatism or fibrositis. The first condition, that of strain, is often wrongly diagnosed as cardiac overstrain. The patient while lifting a weight is suddenly seized with pain over the precordium and he becomes intensely alarmed and thinks that he has strained his heart. If the muscle fibres are very carefully palpated with the tips of the fingers, tender spots or small gaps will be found where the muscle has been torn. From my own experience I would say that the upper attachment of the left rectus and the lower border of the *pectoralis major* are the most common sites where the sore spots are found. This condition generally gets quite well in about three or four weeks.

Fibrositis particularly attacks the intercostal muscles, occasionally the pectorals, and may be easily diagnosed by careful palpation with the tips of the fingers, pressing well into the muscles bit by bit, until fibrous bands, varying in thickness from that of a piece of string to that of half the thickness of a lead pencil, or nodules may be felt. These are intensely tender and the tenderness is confined entirely to these nodules or bands.

We next pass to the ribs and costal cartilages. Remember that there are joints between the sternum and cartilages and the cartilages and ribs, and here we may get rheumatism. Again, careful palpation of these areas will show tenderness over these joints, and there is also pain in them in deep breathing.

In regard to intercostal neuralgia, I cannot remember a single case of this condition which was primary in origin. It is usually due to some other condition, such as spondylitis *et cetera*, which will be mentioned later.

Passing through these structures, the next we come to is the pericardium. Pericarditis, as you are aware, is only a complication of some general disease, particularly rheumatism. The pain here is usually a general one over the precordium and is not of very much importance from our point of view, as the condition is thought of in those diseases where it is likely to occur, and the diagnosis can generally be verified by the presence of precordial friction heard on auscultation. It is well to remember that pericardial pain may be very acute.

Passing from the precordium we come to the heart, and here we have to consider four different types of pain:

1. The general soreness and discomfort which is occasionally met with in cases of endocarditis.
2. *Angina pectoris* only lasting a few seconds or minutes.
3. *Angina pectoris* lasting longer, from minutes to hours.
4. The so-called "false" angina.

General Soreness and Discomfort.

The first condition, that of general soreness or precordial discomfort, as I said before, is occasionally met with in certain cases of endocarditis and cardiac failure. The pain is made slightly worse by exercise, but is not very severe. Examination generally shows a diffuse hyperæsthesia over the precordial region. A condition where there may be pain similar to this, is called, for want of a better name, the "effort syndrome." Here our patients are always neurotic. Their symptoms are out of all proportion to the physical findings. Electrocardiographic examinations in these cases show absolutely nothing abnormal, but their response to effort is often below normal. The conditions are often difficult to distinguish, but if the general aspect of the patient is considered and the whole symptoms and physical findings are added together and compared, the condition will not always present many difficulties in diagnosis. The condition of "effort syndrome" occurs in neurotic young males and in women about the late thirties or a little later.

Pernicious anæmia is a condition which may cause considerable pain in the precordial region on account of anoxæmia of the heart muscle. This condition may easily be mistaken for some cardiac disease on account of the breathlessness of the patient on exertion and the presence of cardiac murmurs. If the physician is aware that pernicious anæmia may set up this trouble, he is not likely to miss it.

Angina Pectoris.

We next come to *angina pectoris*, and the importance of an accurate diagnosis of this condition is essential, because, if the diagnosis is missed and the condition not recognized, the patient will probably be told that he can take exercise greater than his cardiac muscle will allow, with the result of catastrophe to him.

The typical symptoms of *angina pectoris* are pain over the precordium, often shooting down the left arm, but occasionally referred to the right side of the chest or even jaw, and this pain is in the great majority of cases produced by effort, such as walking up a hill. The pain may not come on directly after the effort, but it generally does so. Usually a fear of impending disaster accompanies this pain and the patient has to stop absolutely still and is unable to move. The pain only lasts a few moments. It is of great importance to find out what movements produce this pain, as I have seen a patient with spondylitis affecting the upper thoracic vertebræ get intense symptoms resembling typical *angina pectoris* when he bent his back to the left side or lifted a heavy weight with his left hand, and yet he could easily lift a hundredweight with his right hand when his spine was bent over to the right.

If the patient is not very definite as to what brings about his pain, ask him whether he can walk up hill or make effort after a full meal. This will clinch the diagnosis of *angina pectoris*. The pain radiates down the inner side of the left arm and after a good many attacks there is often hyperæsthesia over the precordial region and along the inner side of the arm. A further test which I found exceedingly useful, is the relief of the pain by the inhalation of amyl nitrite. The condition may be confused with some gastric disturbance. Amyl nitrite

will not relieve a gastric disturbance, but large doses of sodium bicarbonate will. I use this test so frequently that I keep a bottle of "Soda mint" tablets and a box of amyl nitrite capsules on my surgery desk.

Examination in cases of *angina pectoris* is occasionally negative, but generally speaking arteriosclerosis will be found. The patients may be nephritics or diabetics and are generally over forty years of age. *Angina pectoris* does not occur when there are symptoms for cardiac failure, such as dropsy and dyspnœa. It is not found in cases where there is mitral regurgitation or stenosis. The blood pressure may be high or may be normal. It is important to remember that the presence of a low blood pressure in no way excludes *angina pectoris*. Excitement, grief or worry may bring it on. In the great majority of cases electrocardiograph examination will show signs of coronary sclerosis or some grave myocardial disease, but occasionally an electrocardiogram will give no information at all. In these cases diagnosis rests largely on the symptoms and the clinical history of the patient, combined with the relief obtained by the use of amyl nitrite. The pain of *angina pectoris* is intense, differing from most other forms of cardiac pain which accompany cardiac disease.

Angina Pectoris of Coronary Occlusion.

In *angina pectoris* of coronary occlusion we get two types of onset of the cardial pain. Usually the pain is sudden in onset and of great intensity, but it differs from the pain of ordinary *angina pectoris* in that it lasts a considerable time; it may last an hour or even days.

The patient when seized with this condition becomes ashy grey and collapsed, the pulse is rapid and thin, the blood pressure falls, there is even some slight cyanosis. There is usually some slight rise of temperature and some leucocytosis. Physical examination of the heart will reveal weak heart sound and often pericardial friction. If the occlusion takes place slowly, the precordial pain is not so intense at its onset, but it gradually increases in severity, taking half an hour or more to reach its greatest intensity. Electrocardiograph examination reveals a particular type of electrocardiogram in 70% of these patients.

Nitrites do not relieve this pain in any way and the only thing that will relieve it is morphine which may have to be administered in very large doses to get any effect. At times coronary occlusion may set up acute pain in the upper part of the abdomen which may be mistaken for the pain of a perforated gastric or duodenal ulcer or even biliary colic; but the collapse in coronary occlusion is much greater, while the abdominal rigidity is not board-like as when there is a ruptured abdominal organ. The presence of pericardial friction will at once exclude the perforated ulcer or gall stones. After a little while, in these cases of coronary occlusion, the abdominal symptoms will subside and the pain be reverted to the precordial region.

There is a rare type of precordial pain which occasionally occurs in young people suffering from paroxysmal tachycardia. It is sudden in onset and is concurrent with the attack of tachycardia. The pain stops when the tachycardia stops.

So-Called "False" Angina.

Excessive tobacco smoking, particularly of the heavier types of tobacco, and occasionally coffee may set up a pain felt under the upper portion of the sternum. It is not made worse by effort and it has a definite relation to the smoking. It is relieved in a day or two by stopping the use of tobacco, but on returning to the heavier brands of tobacco or even to any smoking, it will come back again.

An excessive gripping pain felt behind the sternum may be met with in digestion, apparently due to hyperchlorhydria. This is often found to be produced by worry, lack of exercise, constipation *et cetera*, and is immediately relieved by large doses of bicarbonate of soda.

Aneurysm of the Aorta.

Aneurysm of the aorta is not met with nearly as often as it used to be. If the coronary circulation is interfered with, the symptoms may resemble those of *angina pectoris*. Physical examination may reveal some of the classical signs of aneurysm, but in the earlier stages it is absolutely impossible to be certain of one's diagnosis of aneurysm of the aorta without the aid of a skiagram. When the aneurysm gets sufficiently large to cause erosion of the sternum, then one will get a peculiar boring, constant pain. The diagnosis will not be difficult in these cases, as the aneurysm will be a considerable size.

Mediastinal Newgrowths.

I cannot remember any case in which mediastinal newgrowth has caused precordial pain, but it is quite possible that it might do so owing to pressure on the intercostal nerves.

Spondylitis.

Spondylitis is one of the most frequent causes of precordial pain and it is in these cases that one will have to cross-question the patient very closely to find out what type of effort makes the pain worse or even brings it about. It may be found that the patient gets pain only when he bends while gardening or, as I mentioned before, when he lifts heavy weights in his right or left hand.

Effort, such as walking up hill or stairs, does not produce pain if the spine is held rigidly. The pain in this condition may be constant or it may come on in paroxysms. There may be no other signs of rheumatoid arthritis in the other parts of the body.

Physical examination will generally reveal some rigidity of the thoracic vertebræ on bending the spine, and there is often tenderness on percussing the spine or attempting to move the individual vertebra with the fingers and thumb.

Always strip these patients' backs quite bare. Scoliosis may at times cause a certain amount of precordial discomfort which may be mistaken for cardiac pain. The pain of scoliosis comes on when the patient is weak or tired, yet if the patient gets fit, he may be able to play golf or tennis without any discomfort at all. Again strip the patient's back and look for sclerosis; if it is present, there will be no difficulty in noticing it.

Tuberculous disease of the spine will be discovered by looking for it. The pain here may be of a nocturnal, darting type.

Tabes may cause precordial pain and so may spinal newgrowths.

Conclusion.

Before leaving off, again let me impress upon you the importance of careful examination and cross-questioning. Strip your patient to the waist and look, listen and feel.

Remember that the great majority of mistakes arise from not looking rather than from not knowing.

MARK C. LIDWILL, M.D.
(Melbourne *et* Sydney),
Honorary Physician, Royal Prince Alfred Hospital, Sydney.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal North Shore Hospital of Sydney on June 12, 1930. The meeting took the form of a series of demonstrations by the members of the honorary staff.

Carcinoma of the Lung.

DR. W. W. INGRAM showed a female patient, aged sixty-nine years, whose illness had commenced six months previously with a cough which was severe and which was accompanied by great pain in the lower part of the left side of the chest. The pain was accompanied by sputum which was white and frothy, but which was not blood

stained. The cough had continued on and off ever since. She had never coughed blood. She had had shortness of breath on exertion since the onset of the illness. The patient suffered from flatulence, dry retching and vomiting, and had epigastric pain. She had lost 12·6 kilograms (two stone) in weight in six months. Since June 1, 1930, she had had very severe pain anteriorly below the left breast, aggravated by coughing. She had lately been constipated. On physical examination the patient appeared slightly anæmic. The chest wall moved but little on respiration, the left side moving less than the right. There was a bulging posteriorly over the right lung due to thoracic scoliosis, with slight convexity to the right. On palpation vocal fremitus was much diminished anteriorly over the left lung and diminished slightly all over the remainder of the chest. On percussion there was pronounced dulness all over the left side of the chest and this was more pronounced anteriorly over the upper lobe. There was dulness posteriorly over the lower lobe of the right lung and the percussion note was slightly impaired anteriorly over the upper lobe of the right lung. On auscultation the breath sounds were absent or distant over the left lung. There was distant tubular breathing below the left clavicle. The breath sounds were vesicular over the right lung and distant posteriorly at the base. The vocal resonance was much diminished anteriorly over the left lung. Elsewhere it was diminished slightly all over the chest. No accompaniments were heard.

There was a firm glandular mass above the medial end of the left clavicle. It was attached to the deeper structures and was not tender to pressure. There was an enlarged gland in the left axilla; it was very firm and not tender. The pulse rate was 90 and regular. The volume, force and tension were normal. The apex beat was not visible, but was palpable ten centimetres (four inches) from the middle line as a weak, circumscribed impulse. The percussion was ill defined, owing to lung dulness. In the mitral area the first sound was impure, ending in a click. The systolic blood pressure was 150 and the diastolic pressure 100 millimetres of mercury. A few pneumococci and streptococci had been found on examination of the sputum, but no tubercle bacilli had been discovered. The other systems were apparently normal. X ray examination of the chest had been undertaken and definite dulness of the left side had been found, suggesting pleural effusion, thickened pleura and/or neoplasm. On aspiration of the chest ten cubic centimetres of turbid yellow fluid had been withdrawn; a second puncture had yielded only a little blood-stained fluid.

Arachno-Dactyly, Rickets and Congenital Pulmonary Stenosis.

Dr. Ingram also showed a child, aged two and a half years, who had weighed 8·1 kilograms (eighteen pounds) at the time of admission. She had been backward from birth, had sat up at ten months, had cut her first tooth at twelve months and had stood up at eighteen months. She had not walked. The child was dark complexioned, had a very placid nature and spoke only a few words. There was hypotonia of all muscles and looseness of tendons and ligaments. The knees could be hyperextended. The ankles, wrists, fingers and toes could be moved far beyond the normal range of joint movement. She had abnormally long phalanges of the fingers and toes. There was slight clubbing of the fingers and the finger tips were bluish. The palate was high arched. The ears were well formed, but had no lobule. The mouth was small and the eyes were set wide apart. The base of the nose was very broad. There was slight bossing of the frontal and parietal bones and the anterior fontanelle was still patent. The patient was pigeon chested and there was a definite Harrison's sulcus. There was a wide subcostal angle. The pulse was regular, but very slow, the rate being fifty-eight. The force was greatly increased and the volume was good. The apex beat was visible in the fourth to seventh spaces and was felt most strongly in the fifth space 5·6 millimetres (two and a quarter inches) from the middle line. The heart dulness was greatly increased. At the mitral area there was a booming first sound with a soft systolic murmur which was not conducted. At the pulmonary area there was a light systolic murmur conducted towards the left shoulder. The second sound was accentuated. The abdomen was pot bellied and the liver edge was palpable several inches below the costal margin. The blood calcium was 8·2 milligrammes and the blood phosphorus was 5·2 milligrammes per hundred cubic centimetres. The diagnosis of the condition was arachnodactyly, slight rickets and congenital pulmonary stenosis.

Tabes Dorsalis.

DR. F. GUY GRIFFITHS showed a woman, aged thirty-seven years, married, who had been admitted to hospital on May 17, 1930, complaining of incessant vomiting and a sharp pain in the left flank of five days' duration. The pain, agonizing in character, had commenced suddenly in the back on the left side and had recurred at frequent intervals ever since. She had been unable to walk ever since undergoing salpingectomy and appendicectomy in October, 1929. There was no history of a rash having appeared on the body. On examination of the nervous system, the knee jerks were absent, the ankle reflexes were absent, the plantar reflexes were flexor in type and the abdominal reflexes were absent. The muscles were flabby and those of the hands exhibited pronounced hypotonus. Rombergism was present. "Point" discrimination was much impaired. The patient's gait was tabetic and she had Argyll-Robertson pupils. She had been given four 0·3 gramme doses of "Novarsenobillon" without any improvement. At the time of the meeting the patient was suffering from another gastric crisis similar to that which had been present at the time of her admission. The Wassermann test yielded a positive reaction and the cerebro-spinal fluid, removed by lumbar puncture, was normal. The patient was suffering from *tabes dorsalis*.

Tuberculosis of the Knee.

Dr. Griffiths also showed a patient who was suffering from tuberculosis of the knee and who had been treated with tuberculin. This case will be reported in a subsequent issue.

Traumatic Partial Pneumothorax.

Dr. Griffiths's third patient was a man who was suffering from traumatic partial pneumothorax. This report will be published in a subsequent issue.

Precocious Syphilitic Paraplegia.

Dr. Griffiths also showed a married woman, aged twenty-one years, who had been admitted to hospital on April 8, 1930, complaining of a "dead" feeling in the leg of two days' duration. She had not passed her urine for thirty hours. The lower part of her abdomen had been swollen and her bowels constipated. She had lost 12·6 kilograms (two stone) in weight during the last three months. She was of spare build. On being asked to move, she could not alter the position of her left leg. General examination revealed no abnormalities except a small fluctuant median tumour in the lower part of the abdomen. On examination of the nervous system, the cranial nerves had been intact. The spinal segments above the twelfth thoracic segment had been intact. The knee jerks had been absent on both sides. The plantar reflex had been extensor in type on the left side and flexor on the right. The ankle reflexes had been absent. There had been no ankle clonus. Motor power had been completely absent in the left lower limb. There had been pronounced weakness of the right lower limb. The limbs had been flaccid and wasted. There had been an absence of light and deep pressure sense in the left leg. The patient had had retention and dribbling overflow of urine which contained pus and the *Bacillus coli communis*. The patient gave a history of syphilis for which she had been treated in the out-patient department for four months. At the time of the meeting she had complete loss of power and sensation in both limbs and complete loss of epicritic and deep sensation from the level of the eighth thoracic segment. There were no abdominal reflexes and the upper limb reflexes were normal.

Dr. Griffiths said that he regarded the patient as suffering from syphilitic meningo-myelitis. She had been given eight doses of "Novarsenobillon" in amounts of 0·2 to 0·3 gramme. On lumbar puncture the cerebro-spinal fluid

had been under increased pressure, but clear, and had shown an increase of globulin. Complement deviation tests for both gonorrhœa and syphilis had failed to yield reactions. The limbs had been placed in light plaster splints to maintain them in good position. It had not been decided whether the abdominal tumour was due to an ovarian cyst or to pregnancy; the "bromine" test had yielded no reaction.

Avulsion of the Phrenic Nerve.

DR. COTTER HARVEY and DR. V. M. COPPLESON showed a series of patients on whom the operation of avulsion of the phrenic nerve had been carried out.

A single woman, aged twenty-one years, had given a history of hæmoptysis for four years. By means of "Lipiodol" bronchiectasis at both bases, but more pronounced on the right side, had been diagnosed. Phrenic avulsion had been performed on the right side and since that time the patient had made steady improvement. For the previous twelve months there had been no hæmoptysis. The patient had gained considerably in weight and was much stronger. X ray examination by the screen showed that the diaphragm was still paralysed. It was pointed out that a definite amelioration of symptoms had occurred.

A married woman had been treated on September 21, 1927, by artificial pneumothorax for severe right-sided pulmonary tuberculosis. On July 24, 1929, treatment had been abandoned on account of obliteration of the pneumothorax cavity. On October 2, 1929, right phrenic avulsion had been carried out and the patient's condition had been maintained ever since. It was pointed out that the indication for phrenic avulsion had been obliterative pneumothorax in pulmonary tuberculosis to reduce permanently the volume of the right lung. The result was very satisfactory.

A man, aged twenty-nine years, had been admitted on March 26, 1929, with a pulmonary abscess towards the base of the right lung. He had been treated by artificial pneumothorax for approximately six months with apparent cure. The lung had been allowed to reexpand, the symptoms had recurred. A further attempt at pneumothorax had been unsuccessful. On December 18, 1929, avulsion of the right phrenic nerve had been carried out. Seen one month before the meeting, the patient had looked very well. There were no further symptoms and the right diaphragm was still paralysed. The patient was regarded as cured.

A male patient, aged fourteen years, had been first seen on May 25, 1928. His condition had been diagnosed as an empyema of the left side following pneumonia six weeks previously. An empyema had been drained and had healed two months later. Several breakdowns had occurred during the following six months. On February 11, 1929, a diagnosis of pulmonary abscess, secondary to old empyema, had been made, and on thoracotomy neither abscess nor empyema had been discovered. On "Lipiodol" examination, bronchiectasis of the left base had been diagnosed and phrenic avulsion had been performed. Since then the patient had improved greatly and gained 12·6 kilograms (two stone) in weight. He had lost all symptoms of toxæmia and both cough and sputum had been considerably reduced.

Thyreotoxic Goitre.

DR. ERASMUS BLIGH showed a male patient, aged twenty-three years, who had been admitted to hospital on April 18, 1930, with a history of nervousness, tremor and palpitation and swelling in the region of the thyreoid gland since he was eleven years of age. Partial thyreoidectomy had been performed when he was eleven, but the symptoms were still present. On May 7, 1930, a further partial thyreoidectomy had been undertaken.

Gastric Ulcer and Hour Glass Stomach.

Dr. Bligh also showed a patient, aged forty-six years, who had been admitted on February 4, 1930, complaining of epigastric pain of many years' duration which had been much worse during the previous six months and which had no definite relation to food. The patient had had persistent vomiting for six months as well as attacks of alternating constipation and diarrhœa. Extreme loss of weight had occurred. On X ray examination, hour glass contraction of the stomach had been discovered with pronounced delay of the meal in the second part. A great deal of the meal had been found in the stomach at the end of seven hours. It had been thought that the constriction was probably secondary to ulcer in the lesser curvature, but no actual crater had been discovered. At operation two definite constrictions had been found, with ballooning of the duodenum. Partial gastrectomy had been performed and both constricted areas had been removed. On pathological examination of the specimen, two healed ulcers of the stomach with considerable cicatrization had been discovered, but with no sign of malignant disease. The patient made an uninterrupted recovery.

Multiple Secondary Carcinomata.

DR. E. MORRIS HUMPHERY showed a female patient, aged thirty-two years, who was unmarried. In November, 1929, her left breast had been removed for carcinoma. At that time the patient had complained of aching pains in both legs. She had been admitted again on April 17, 1930, complaining of the same pain and of inability to walk. On examination a hard, round tumour of the right frontal bone was present and the scar of the previous operation was breaking down. No glands were palpable in axillæ, groins or neck. Radiological examination revealed extensive metastases of the lumbar spine and pelvis, almost certainly carcinomatous. The appearance was suggestive of Paget's disease of the dorso-lumbar spine. Extensive carcinomatous metastases were present throughout the right frontal and parietal regions. The lungs were apparently clear.

Fracture of the Tibia.

Dr. Humphery also showed a patient, aged twenty years, who had suffered a severe compound fracture of the left tibia in October, 1928. Splints had been applied and the patient had been discharged with the leg in plaster in March, 1929. On X ray examination a small amount of bony callus had been found. In September, 1929, the fracture had been still ununited and the tibia had been wired. Following operation the wound had persistently discharged and several small sequestra had been extruded. X ray examination in November, 1929, had revealed an area of rarefaction round the wire and the wire had been removed. The wound had healed and the patient had been discharged in December, 1929, with apparently slight union. He had been readmitted in May, 1930, with fibrous union only. Open operation had been performed, fibrous tissue removed, the ends of the bones freshened, the wound closed and plaster fixation applied.

Low Cæsarean Section.

DR. OSSIAN ROBERTSON showed a female patient, aged twenty-three years, who had been submitted to low Cæsarean section in her first pregnancy. The patient's measurements were as follows: Interspinous, 20 centimetres (eight inches); intercristal, 25 centimetres (ten inches); external conjugate, 16·8 centimetres (six and three-quarter inches). The transverse diameter of the outlet was 5·6 centimetres (two and a quarter inches) and the post-sagittal measurement 5·6 centimetres. Dr. Robertson said that the low Cæsarean section operation had been found by him specially applicable to patients in whom interference had taken place and in whom delivery by forceps had been attempted, as well as those in whom the classical operation would probably be fatal.

Fracture of the Femur.

DR. V. M. COPPLESON showed a patient, aged forty-seven years, on whom he had performed Whitman's operation for fracture of the neck of the femur. The patient had been admitted in October, 1929, with an unimpacted fracture of the neck of the femur. As no bony union had occurred, Whitman's operation had been carried out in February, 1930. At the time of the meeting the patient was walking fairly well on crutches.

Sinusitis.

DR. E. P. BLASHKI showed two patients, one of whom had suffered from fulminating frontal sinusitis and the other from double mastoiditis following scarlet fever. The two cases will be reported in a subsequent issue.

Osteochondritis Dissecans.

DR. S. H. SCOUGALL showed a male patient, aged nineteen years, who was suffering from *osteochondritis dissecans*. This case will be reported in a subsequent issue.

Osteochondromatosis.

Dr. Scougall also showed a male patient, aged forty-five years, who was suffering from osteochondromatosis. He showed this patient in order that the condition might be compared with that of the patient suffering from *osteochondritis dissecans*. In the latter condition loose bodies in the knee had been formed by traumatic detachment of a portion of the articular surface. The patient with osteochondromatosis had an osteoarthritis of both knees and multiple loose bodies were present in the joint cavity. These synovial chondromata occurred quite distinctly from detached osteophytes and in this instance were diffuse. Those of the diffuse type were generally free, while the single and multiple types might be pedunculated or free. Dr. Scougall said that Jones and Lovett described three groups of symptoms according to the attachment and location: (i) A portion of articular surface was completely detached, wandered about and became secondarily attached to the synovial membrane; (ii) the detached piece became immediately adherent and ultimately detached; (iii) gradual, slow detachment occurred.

Osteomyelitis of the Mandible.

DR. H. HUNTER JAMIESON showed a male patient, aged four months, who had been admitted with a history of ulcers in the mouth for two weeks. During this time the patient had been fed with half malt and half "Lactogen"; prior to this the child had been fully breast fed. On examination the whole of the right side of the face had been swollen and as a result of pressure pus had been evacuated from the inside of the cheek in the region of the parotid duct. The face had been incised over the fluctuant area and much pus had been evacuated. On X ray examination extensive osteomyelitis of the mandible had been found on both sides. Eighteen days later an incision had been made on the right side of the lower jaw and several sequestra had been removed. A month later the same operation had been performed on the left side. Dr. Jamieson pointed out that the child's condition had improved considerably and that the teeth on appearance were not irregular.

Varicose Veins.

Dr. Jamieson also showed several patients whose varicose veins had been treated by the injection of a quinine-urethane solution. The results were satisfactory.

THE ANNUAL GENERAL MEETING OF THE SECTION OF CLINICAL MEDICINE OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Adelaide Hospital on August 20, 1930.

The report for the year was presented. The membership of the Section is now fifty-two. Four meetings were held during the year, with an average attendance of thirty members.

The following officers were elected:

Chairman: Dr. E. Britten Jones.

Members of Committee: Dr. R. L. Thorold Grant, Dr. F. Ray Hone, Dr. Guy Lendon.

Honorary Secretary and Treasurer: Dr. A. R. Southwood.

A clinical meeting followed, and the following members demonstrated cases: Dr. Mildred Mocatta, Dr. Ray Hone, Dr. F. S. Hone, Dr. John Close, Dr. A. R. Southwood.

THE JUBILEE OF THE NEW SOUTH WALES BRANCH.

THE New South Wales Branch of the British Medical Association was founded in 1880. It celebrates its jubilee this year. The occasion will be commemorated on Friday, October 3, 1930. The new house of the Branch at 135-137, Macquarie Street, Sydney, will be opened officially by His Excellency Sir Philip Game, Governor of New South Wales, at 3 o'clock p.m. on October 3, and an official dinner will be held in the evening at the University Club.

The time of the ceremony has been well chosen. During the same week Sydney will be the rendezvous of members of the medical profession from all parts of Australia. The Federal Committee of the British Medical Association in Australia meets in Sydney during that week; there will also be the annual general meeting of the Australasian Medical Publishing Company Limited. In addition, many Fellows of the College of Surgeons of Australasia will be attending a meeting of the College. Invitations have been sent to officers of the several Branches in other States. The gatherings will thus be representative of the whole Commonwealth.

Admission to the afternoon ceremony will be by ticket only and each ticket will admit a member and one lady. After the ceremony there will be an inspection of the house. Refreshments will be served. Academic dress or full morning dress will be worn. The official dinner will be held at 7 p.m. on Friday, October 3, 1930, at the University Club, Phillip Street, Sydney. The guests to be invited will include His Excellency the Governor, the Honourable the Premier, the Honourable the Minister for Health, the Chancellor and Vice-Chancellor of the University and the Lord Mayor of Sydney.

Tickets for both ceremonies may be obtained by members from the Honorary Secretary of the Branch, 135-137, Macquarie Street, Sydney.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, numbers 49, 51, 56 and 65, of June 12, 19, July 3 and 31, 1930.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Promotion.—Surgeon Lieutenant Lionel Lockwood is promoted to the rank of Surgeon Lieutenant-Commander, dated 12th May, 1930.

Confirmation in Rank.—Surgeon Lieutenants (on probation) William George Farrell and Henry Kemple Byron Bailey are confirmed in the rank of Surgeon Lieutenant, with seniority in rank of 1st June, 1929.

Promotions.—Surgeon Lieutenant-Commander David Shields Prentice is promoted to the rank of Surgeon Commander, dated 1st July, 1930.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve (Sea-Going).

Promotion.—Surgeon Lieutenant-Commander Harold Henry Field-Martell is promoted to the rank of Surgeon Commander, and is re-appointed, temporarily, as District Naval Medical Officer, Fremantle, dated 7th June, 1930.—(Ex. Min. No. 92.)

AUSTRALIAN MILITARY FORCES.

First Military District.

Australian Army Medical Corps.

Lieutenant L. A. Little is transferred from the Melbourne University Rifles, 4th Division, and to be Captain (provisionally) supernumerary to the establishment pending absorption, 23rd April, 1930.

Captain G. F. Cherry is supernumerary to the establishment pending absorption, 1st July, 1930; Major C.

Shellshear is transferred to the Australian Army Medical Corps Reserve, 1st July, 1930. The provisional appointment of Captain J. H. Blackburn is terminated, 30th June, 1930, and he is transferred to the Australian Army Medical Corps Reserve, and to be Honorary Captain, 1st July, 1930.

Second Military District.

First Cavalry Division: Staff.

Lieutenant-Colonel P. Fiaschi, O.B.E., V.D., Australian Army Medical Corps, is appointed Assistant Director of Medical Services and is granted the temporary rank of Colonel whilst holding such appointment, 1st July, 1930, *vice* Colonel J. C. Storey, O.B.E., V.D., who relinquishes the appointment on 30th June, 1930.

Australian Army Medical Corps.

Pharmaceutical Service—To be Captain: Lieutenant W. G. Masters, 8th May, 1930.

To be Captains (provisionally)—Lieutenant N. P. Breden, Lieutenants (provisionally) F. W. Clements, F. B. Uther, G. G. L. Stening, and G. E. Phillips, Lieutenants W. N. Newton and L. E. Odlum, and Lieutenant (provisionally) H. M. Rennie, 1st July, 1930. Captains (provisionally) F. B. Uther, G. G. L. Stening, G. E. Phillips, W. N. Newton, L. E. Odlum, and H. M. Rennie to be supernumerary to the establishment pending absorption, 1st July, 1930. Colonel J. C. Storey, O.B.E., V.D., is transferred to the Unattached List, 1st July, 1930. Lieutenant-Colonel P. Fiaschi, O.B.E., V.D., relinquishes the command of the 4th Cavalry Field Ambulance, 30th June, 1930.

Major W. Vickers, D.S.O., V.D., from the command of the 2nd Field Hygiene Section, is appointed to command the 4th Cavalry Field Ambulance, 1st July, 1930; the provisional appointment of Captain L. E. Odlum is confirmed.

Australian Army Medical Corps Reserve.

Lieutenant-Colonel R. G. Craig is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 24th May, 1930; Honorary Captains W. B. Kerr, J. McMaster and E. Jeffrey are retired, 28th May, 1930.

Honorary Captains F. H. Cox and O. R. P. Muller are retired, 18th June, 1930, and 30th June, 1930, respectively.

Honorary Captain W. J. Chapman is retired.

Award of the Colonial Auxiliary Forces Officers' Decoration.

Australian Army Medical Corps—Lieutenant-Colonel W. E. Kay, D.S.O.

Third Military District.

Australian Army Medical Corps.

The provisional appointment of Captain E. G. Robertson is terminated, 6th May, 1930, and he is transferred to the Australian Army Medical Corps Reserve, and to be Honorary Captain, 7th May, 1930; the notification of the transfer to the Australian Army Medical Corps Reserve of Lieutenant J. Bastow, which appeared in Executive Minute No. 56/1930, promulgated in *Commonwealth Gazette*, No. 33, of 1st May, 1930, is cancelled.

Major T. G. S. Leary is appointed to command the 15th Field Ambulance, 24th June, 1930. *To be Captain (provisionally)*—Lieutenant E. J. Grieve, 1st July, 1930.

To be Lieutenant (provisionally) supernumerary to the establishment pending absorption—Henry James Bondfield Stephens, 1st June, 1930. Lieutenant-Colonel R. C. Withington relinquishes the command of the 15th Field Ambulance, 23rd June, 1930, and is transferred to the Australian Army Medical Corps Reserve, 24th June, 1930; the provisional transfer from the Melbourne University Rifles, 4th Division, of Lieutenant H. D. Phipps is terminated, 30th June, 1930, and he is transferred to the Australian Army Medical Corps Reserve, 5th Military District, and to be Honorary Captain, 1st July, 1930.

The provisional appointments of Captain F. S. Loughnan, E. L. Cooper and G. A. M. Knight and Lieutenants T. G. B. Allen and M. G. Edison are confirmed. Captain G. A. M. Knight is seconded, 1st July, 1930.

Captain E. L. Cooper is brought on the authorized establishment, 1st July, 1930.

Australian Army Medical Corps Reserve.

Major (Honorary Lieutenant-Colonel) J. C. Morton, V.D., is placed upon the Retired List, with the honorary rank of Lieutenant-Colonel, and with permission to wear the prescribed uniform, 23rd June, 1930; Captain J. G. Desailly is retired, 21st June, 1930.

Major C. H. Anderson is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 7th July, 1930. Honorary Major C. R. Player is retired, 2nd July, 1930.

Fourth Military District.

Australian Army Medical Corps Reserve.

Lieutenant Colonel A. M. Cudmore is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform 11th June, 1930.

Honorary Lieutenant C. Webb is retired, 29th July, 1930.

Fifth Military District.

Australian Army Medical Corps.

Captain M. F. Williams is appointed (provisionally) from the Australian Army Veterinary Corps Reserve and is supernumerary to the establishment pending absorption, 7th June, 1930.

Australian Army Medical Corps Reserve.

To be Honorary Captains.—Harry Fenwick Hustler and Honorary Lieutenant S. F. Sutherland, 7th June, 1930, and 1st July, 1930, respectively. *To be Honorary Lieutenant.*—Ralph Joseph Farnbach, 3rd June, 1930.

To be Honorary Lieutenant—Henry Markham Trethowan, 21st May, 1930.

To be Honorary Captain—Douglas Duncan McCowan, 18th June, 1930.

Sixth Military District.

12th Mixed Brigade: Infantry.

40th Battalion.—Honorary Captain N. M. Simpson is appointed from the Australian Army Medical Corps Reserve and to be Lieutenant (provisionally), 30th May, 1930.

Correspondence.

OTITIS MEDIA.

SIR: The excellent epitome of the subject of acute *otitis media* by Dr. Robert Godsall in the journal of this date is lacking in one very important particular. The omission is one that is common to modern papers on surgical subjects—the dressing and after-care of wounds.

Whatever Dr. Godsall's technique in this regard may be, he has left us in the dark. The vital importance of guarding against secondary infection in the discharge following paracentesis for acute suppuration of the middle ear cannot be disregarded and invariably failure to prevent this complication is the cause of persistence of otorrhœa and not necessarily because "the mastoid is too deeply involved." My suggestion is to paint the meatus with soft "Bipp" and cover the auricle with any kind of antiseptic dressing to receive the discharge. No plugging on any account. Acute cases treated in this manner invariably show only a rapidly diminishing serous discharge after forty-eight hours and, inflation as usual, a cure results within eight or ten days. Pus from any source, especially at a raised body temperature, is an ideal culture medium, particularly for the ubiquitous proteolytic agents. It astounds me that no provision is suggested against such harmful changes.

Yours, etc.,

A. C. F. HALFORD.

Wickham House,
Wickham Terrace,
Brisbane.
August 30, 1930.

NOTICE.

THE Registrar of the University of Melbourne has asked us to announce that the Peninsular and Oriental Steam Navigation Company, Limited, has vacancies for assistant surgeons on the R.M.S. *Moldavia* (October 7) and also on the R.M.S. *Mongolia* (November 4). Applications for these positions from graduates of the University of Melbourne will be received by the Dean of the Faculty up to September 20, 1930.

Books Received.

GONOCOCCAL INFECTION IN THE MALE, by A. L. Wolbarst, M.D.; Second Edition, Revised and Enlarged; 1930. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay. Royal 8vo., pp. 297, with 140 illustrations, including 7 colour plates. Price: $5.50 net.

A TEXTBOOK OF MASSAGE FOR NURSES AND BEGINNERS, by Maude Rawlins; 1930. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay. Post 8vo., pp. 144, with illustrations. Price: $2.00 net.

Diary for the Month.

SEPT. 16.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 17.—New South Wales Branch, B.M.A.: Section of Obstetrics and Gynæcology.
SEPT. 17.—Queensland Branch, B.M.A.: Eye, Ear, Nose and Throat Section.
SEPT. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 24.—Victorian Branch, B.M.A.: Council.
SEPT. 25.—South Australian Branch, B.M.A.: Branch.
SEPT. 25.—New South Wales Branch, B.M.A.: Branch.
SEPT. 26.—Queensland Branch, B.M.A.: Council.
OCT. 1.—Victorian Branch, B.M.A.: Branch.
OCT. 2.—Federal Committee of the B.M.A. in Australia.
OCT. 2.—Queensland Branch, B.M.A.: Council.
OCT. 3.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates of Local Associations with the Council.
OCT. 3.—Queensland Branch, B.M.A.: Branch.
OCT. 7.—New South Wales Branch, B.M.A.: Council.
OCT. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments.

Dr. A. C. Blumer (B.M.A.) has been appointed Government Medical Officer at Trundle, New South Wales.

* * *

Dr. M. Jacobs (B.M.A.) has been appointed Certifying Medical Practitioner at Bendigo, Victoria, under the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. G. H. Guthridge (B.M.A.) has been appointed Certifying Medical Practitioner at Yarraville, Victoria, under the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. V. H. Webster has been appointed District Medical Officer and Resident Magistrate, Protector of Aborigines (until December 31, 1930) and Quarantine Officer (under the provisions of the *Quarantine Act*, 1908-1924) at Wyndham, Western Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xiv.

ADAVALE HOSPITALS BOARD, QUEENSLAND: Medical Officer.
BURREN JUNCTION MEDICAL COMMITTEE: Medical Officer.
ECHUCA DISTRICT HOSPITAL: Resident Medical Officer.
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Vacancies.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital. Boonah Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.